ABSTRACT

OF

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An Abstract of the Proceedings

OF THE

Association of Life Insurance Medical Directors of America

FORTY-FIFTH ANNUAL MEETING

The Forty-fifth Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Waldorf-Astoria Hotel, New York City, on October 18 and 19, 1934.

The following members, delegates and guests were present at some time during the sessions: Doctors J. W. Abbott, G. E. Allen, C. D. Alton, H. H. Amiral, H. B. Anderson, E. M. Armstrong, T. M. Armstrong, W. B. Aten, A. E. Awde, B. M. Baker, G. H. Barber, N. J. Barker, W. B. Bartlett, J. T. J. Battle, E. W. Beckwith, J. E. Bee, M. B. Bender, J. R. Biggs, C. C. Birchard, W. F. Blackford, D. N. Blakely, H. E. Bogart, J. E. Boland, William Bolt, E. C. Bonnett, J. T. Bowman, F. G. Brathwaite, C. T. Brown, H. B. Brown, B. F. Byrd, A. C. Cameron, E. J. Campbell, J. H. Campbell, F. H. Carber, P. E. Carlisle, H. R. Carstens, L. B. Cavins, C. L. Christiernin, H. W. Cook, D. B. Cragin, B. B. Crohn, R. M. Daley, H. D. Delamere, E. J. Dewees, E. G. Dewis, H. K. Dillard, E. S. Dillon, C. R. Dudley, J. H. Dunkley, O. M. Eakins, F. C. Evers, J. G. Falconer, R. K. Farnham, H. H. Fellows, W. E. Ferguson, W. S. Fewell, R. S. Fowler, R. A. Fraser, E. M. Freeland, H. M. Frost, F. I. Ganot, E. E. Getman, R. A. Goodell, J. K. Gordon, R. J. Graves, A. H. Griess, F. L. Grosvenor, F. W. Hag-

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Total attendance at all sessions, 194.

During the past year, four members of our Association have died. The following testimonials to their memory have been prepared by representatives of the Association.

DR. WILLARD B. CARPENTER

Dr. Willard B. Carpenter, Vice President of The Columbus Mutual Life Insurance Company, Columbus, Ohio, died September 19th. He had been in ill health for months. Two operations were performed in the hope of saving his life.

For nearly 26 years Dr. Carpenter served faithfully as Medical Director of the Columbus Mutual, retiring in January, 1934. Due to his discriminating selection of risks the Company established a notably low mortality record.

Dr. Carpenter's work as Medical Director was characterized by conscientious and painstaking care. He was rightfully proud of his Department's record.

Joining C. W. Brandon, the Company's Founder, in promoting the Company in 1906, Dr. Carpenter was Secretary as well as Medical Director when the Company began business in 1908. During the Company's first years, growth was slow but steady. Dr. Carpenter shared with Mr. Brandon a vision of future achievement, and lived to see the Company nationally recognized as worthy of a place among the leaders.

Gracious and kind, Dr. Carpenter was highly esteemed by his office associates and by Fieldmen. All had warm admiration for his numerous fine qualities.

Born in Ross County, Ohio, nearly 79 years ago, Dr. Carpenter was the son of a Presbyterian minister, and he continued a follower of that faith throughout his life. While a young man he was a Sunday School teacher.

Dr. Carpenter was educated at Wooster College, Wooster, Ohio, and at Hahnemann Medical College, Philadelphia, Pa. While in Philadelphia he became a member of the Black Horse Troop, a famous equestrian organization of Masons. Later he joined the Odd Fellows.

For over 55 years Dr. Carpenter practiced Medicine in Columbus. As a family physician he was widely known and beloved. Children at whose birth he officiated have become citizens of prominence in his home city. Dr. Carpenter conducted a private hospital for some years in Columbus.

A man of choice tastes, Dr. Carpenter was interested in literature and music. In earlier years he played the organ in two Columbus churches. In 1876 he performed on the organ at the nation's Centennial celebration in Philadelphia.

Dr. Carpenter's wife, Mrs. Margaret Fisher Carpenter, to whom he was married Thanksgiving Day, 1933, survives him.

Dr. Carpenter became a member of the Association of Life Insurance Medical Directors in 1928.

DR. JOHN CHARLES HUMPHREYS

It is with sincere regret that I bring to your attention the death, on March 31, 1934, of Doctor John Charles Humphreys, Director Health Bureau, The Penn Mutual Life Insurance Company, and a member of this Association.

Doctor Humphreys was born in Philadelphia on July 26, 1879. He received his preliminary education in the Philadelphia public schools and later graduated from the historic Germantown Academy. He entered the Hahnemann Medical College, graduating with the Class of 1908. After serving an interneship in the Hahnemann Hospital, he spent a year in further study at Jefferson Medical College, securing a degree from that Institution in 1910.

The next fifteen years, from 1910 to 1925, Doctor Humphreys spent in medical missionary work in West China for the American Baptist Foreign Missionary Society, with but two years' leave of absence in this country—from 1917-19. His devotion to his work and the sincerity of his interest in his chosen profession are illustrated by the fact that he devoted his entire leave of absence to public speaking in behalf of the Society and in post-graduate work in the clinical laboratories of the Post-Graduate School of the University of Pennsylvania.

The Doctor's experiences in China covered a wide range of interests. He labored consistently to promote the sanitary and general health conditions of China; and, together with a representative of the Chinese Health Council, he inaugurated the first big health program and campaign in West China. He also inaugurated the practice of periodic health examinations for the student body of West China Union University, at Chengtu, of which university he was a member of the Faculty of Medicine.

For his devotion to his work and his untiring efforts in ministering to the people of West China, regardless of creed or color, he received international recognition. The Chinese Government conferred upon him the Red Cross Award of Honor for the unselfish service he rendered in famine relief; and the French Government awarded him special recognition for his care of French citizens and government officials.

Early in 1925, he had the first indications of a heart impairment, which forced him to give up his career as a medical missionary, and, nine years later, was to cause his death; yet, soldier that he was, he never at any time alluded to this condition.

He came with The Penn Mutual Life Insurance Company on July 12, 1926, having spent the greater part of the intervening years in post-graduate study in Preventive Medicine and Health Conservation at Columbia University and at Jefferson Medical College.

To his work with The Penn Mutual he brought the same sincerity of purpose and whole-heartedness of effort which characterized his every activity and won at once the confidence of all who came in contact with him.

In addition, he continued to lecture on his work in China and to give unsparingly of his time and energies to religious activities, in which he ever remained deeply interested.

His home life was an ideal and an exceptionally happy one, shared by his devoted wife and their son and daughter, who survive him.

He was ever gentlemanly, courteous, kindly, and helpful; a faithful officer, a sincere friend, and a loyal business associate. Those of us in daily touch with him will greatly miss his wholesome and helpful companionship.

DR. HENRY SEATON HUTCHISON

It is with the deepest of regret that I bring to your attention the death of Dr. Henry Seaton Hutchison, Chief Medical Officer of the Crown Life Insurance Company of Toronto, and a member of the Association.

Dr. Hutchison was born in Toronto in 1879, the son of the late Henry Hutchison of the publishing firm of Rowsell and Hutchison. He received his early education at the Model School, Toronto, and at Upper Canada College. He graduated in Medicine from the University of Toronto in 1900, later serving as an interne both in the Hospital for Sick Children and in the Toronto General Hospital. In 1903 he was appointed an assistant in the clinical laboratories of the Department of Pathology and served as Demonstrator in Pathology from 1906 to 1908. He joined the staff of the Department of Medicine of the University of Toronto as an Assistant in 1907, the same year being appointed a Junior Attending Physician to St. Michael's Hospital. He resigned from the staff of this hospital in 1915 and was later appointed an Attending Physician to the Toronto General Hospital. He was made an Associate in Medicine in 1922 and in 1929 was appointed Physician-in-Charge of the Out Patient Medical Clinic of the Toronto General Hospital and Assistant Professor of Medicine in this Faculty. He was a Charter Fellow of the Royal College of Physicians of Canada.

Dr. Hutchison, for many years, was very interested in the insurance medicine as an examiner and for a number of years was on the staff of the Canada Life Assurance Company of Toronto. In 1928 he was appointed Associate Medical Officer for the Crown Life Insurance Company of Toronto and in 1930, following the death of the late Dr. Henry Machell, became Chief Medical Officer.

His interests were not confined to his chosen profession for he was a lover of good music and good literature but his busy professional life called for the sacrifice of many personal interests and he gave it uncomplainingly. He possessed an unusual capacity for friendship and evinced a keen appreciation of talents in others.

His sudden death in the midst of a busy day was a shock that will linger for many a day in the hearts of his patients and friends. His quiet presence and considered speech will be greatly missed by all those who have been associated with him.

DR. PHINEAS H. INGALLS

Death ended the brilliant career of Dr. Phineas H. Ingalls, December 14, 1933. Thirty-three of the fifty-three years in which he engaged in medicine he was associated with the Ætna Life Insurance Company. He came with us as Associate Medical Director in January, 1900, and served in this capacity until his death.

Dr. Phineas H. Ingalls was born in Gorham, Maine, in 1856, the son of Dr. Phineas Ingalls who was the leading physician in this town. He graduated from Bowdoin College in 1877, receiving a Master of Arts degree in 1883. He studied medicine with Drs. Tooksbury and Charles W. Bray of Portland and completed the course at the College of Physicians and Surgeons in New York, graduating in 1880.

In 1881 he came to Hartford, succeeding the late Dr. Matthew W. Mann. He was appointed gynecologist of the Hartford Hospital in 1884 and until 1922 he was in active surgical practice, visiting the hospital daily and performing all the operations pertaining to his specialty. In addition to the stress and strain of an active surgical practice, it must not be forgotten that this was during the period of the new developments in surgery which practically involved a new technique and a new viewpoint of all surgical procedure.

In 1922 his health was beginning to fail and he closed his office and retired from the practice of his specialty, but he still continued his active service in the hospital, retaining chairmanship of the various committees on which he served, until 1933 when he could no longer act because of ill health.

One element in the long service Dr. Ingalls gave to the profession as well as to the hospital does not appear in any formal report or resolution but is well known and fully appreciated by

many of the medical profession. For years a succession of house physicians came under his care, instruction, and tutelage. It is not too much to say that the succeeding group of young men who were taught by him not only operative technique, the routine care of convalescents after operation, but best of all, they learned from him the balancing of judgment necessary to decide when surgery was essential, when it could be postponed, and when it should not be attempted. These things are not easily obtained from books and best obtained by experience and tutelage under an able and observant and superior officer. It is not too much to say that the influence Dr. Ingalls had and the instruction he gave over his succeeding house officers were the leading factors in the professional success which so many of them have since obtained.

As a member of our medical department, Dr. Ingalls was alert to new developments, untiring in his devotion to the fast-increasing duties of the medical staff, and invaluable for his clear, well balanced judgment on problems coming before the medical department for decision. Earlier in his association with us he did some traveling in the field. Until more recent years he attended field conferences regularly. The field organizations came to know him well, to hold him in high and enthusiastic esteem.

Always friendly and approachable, a splendid companion, and a wise counsellor, we mark his passing with profound regret.

President Fraser in the Chair.

Dr. Fraser—Gentlemen, the forty-fifth annual meeting of the Association is now called to order. I notice on the first order of the meeting that the Secretary has placed before me "Opening Remarks of the President". I hope you will forgive me if I do not make any real opening remarks, as unfortunately an attack of laryngitis prevents any extended remarks by the Chair. I am hopeful that we may all derive benefit from the papers that are to be presented before us and I wish to extend to you my thanks for your presence here today.

I am sorry to state that our good friend, Dr. Hunter, Vice President and Actuary of the New York Life Insurance Company, who has always been pleased to be present at our meetings, will not be with us today. An unfortunate circumstance arose, due to which the meetings of the Actuarial Society and the Medical Directors Association take place on the same day—the Actuarial Society meeting in Washington. Dr. Hunter felt that it was incumbent upon him to be present at that meeting.

Our good friend, Dr. Rogers, whom I saw a few days ago, told me that he doubted if he could be present, although he did wish to have his remembrance carried before you all.

Dr. Angier Hobbs also wished to have the expression of his good wishes placed before you.

I regret to state that there have been some changes in the program which has been submitted to you. Dr. Arlie R. Barnes, Chief of the Cardiac Section of the Mayo Clinic, was to present the paper on "Coronary Sclerosis as an Insurance Problem". At the last moment Dr. Barnes found that he had to leave for Europe. We will be unfortunate in not having him here to take part in the final discussion of his paper and I ask you all to read his paper so that when the discussers take their part in the program, you will understand the message Dr. Barnes intended to convey.

Also noted on the program was the fact that Dr. Henry F. Starr, of the Pilot Life Insurance Company, was to take part in the discussion of Dr. Wesley Scott's paper on systolic murmurs. Dr. Starr was unfortunately taken quite ill, but I believe he is now convalescent. I am sorry that he will not be able to take his part in the program.

To fill in the vacancies caused by the absence of these two gentlemen, Dr. Hunter has very kindly consented to present discussions on Dr. Scott's paper on systolic murmurs and also on the paper on blood pressure submitted by Dr. William Bolt. These discussions will be presented by the Chair.

DR. FRASER—We have with us today one of the chief executives of the New York Life Insurance Company, who will pre-

sent the address of welcome to the delegates at this convention. I have much pleasure in introducing to you Mr. L. Seton Lindsay, our First Vice President of the New York Life Insurance Company. (Applause.)

MR. LINDSAY—Dr. Fraser and Gentlemen of the Medical Profession: It is a great pleasure to welcome you to New York and I take it as a peculiar compliment that your President and our Chief Medical Director, Dr. Fraser, should have selected an agency officer to welcome you to New York. I can imagine the remembrances some of the elder men here possibly may have of the horror that the Medical Department had of the Agency Department some twenty-five years ago when I was starting in, and this compliment your Chairman has given me is perhaps symbolic of the better relations between Medical men and Agency men generally.

I think misunderstanding usually comes because one is not fully acquainted with the facts. Years ago when I was a young agent in the field I know that if I had a chap that weighed three hundred pounds, I looked around until I could find—well, Dr. Fraser would be the sort of Medical Director I would choose (laughter) or would like to put that case before; we would play one Medical Director against another.

Now with your scientific rating of risks, that is not possible any longer, and good feeling between us exists, I think that you feel that we don't try to put things over on you and I know very well that you don't try to put things over on us.

I might tell you a short story about a misunderstanding of the facts which I think may make clear my point about the misunderstandings in the old days between agents and doctors. One of these fellows who was very conscious of his own rights was riding in a street car and the conductor came along and said: "Take that bag off the seat." He said: "No, I don't want to take the bag off the seat." "Take it off," said the conductor, and this fellow replied, "Take it off yourself." The conductor said: "I am going up to the end of the aisle here and if you don't have the bag off the seat by the time I get back, I'll throw it out the window."

Well, he walked up to the end of the car and when he came back, the bag was still there. So he said: "Why didn't you put the bag off the seat? Didn't I tell you I would throw it off the car if you didn't?" The other replied: "I didn't think you would do it." Well the conductor picked up the bag and threw it out of the window. The passenger said: "I never thought you would do it." The conductor replied: "Well, I warned you." The other then said: "Oh, that's all right, it really doesn't matter to me because it isn't my bag." (Laughter.) That's what I mean by not knowing the facts.

Gentlemen, I don't want to take up too much of your time, but these are days of cooperation. I think that is the great spirit abroad everywhere and the cooperation between the Medical Directors and the Actuaries and the Agency Department as well, I think, is typical of our business. I often wonder if the cooperation between life insurance companies and between individuals in life insurance companies today doesn't really point perhaps a good example for other business. We are all cooperating under what might be called the mutual principle, and I believe that this cooperative spirit is very well shown in your organization and that of the actuaries and is a very helpful sign. We all ought to be happy that we are in the life insurance business instead of some other business. (Applause.)

DR. FRASER—I want to thank you, Mr. Lindsay, for your kindness in appearing before us and also for the expression of cooperation which exists between the Medical Department and the agency force at our Home Office.

This concludes the business meeting of this convention. I note that again the Secretary has put in the order of procedure "Opening Remarks by the President". We will dispense with any further remarks.

Secondly, "Remarks by Executive of the New York Life Insurance Company". Mr. Lindsay has already appeared before us and made a splendid opening address of welcome.

Thirdly, "Remarks by Dr. Joseph E. Daniel, Medical Director of the Great Southern Life Insurance Company and Chairman

of the Medical Section of the American Life Convention". I received a telegram from Dr. Daniel stating that it would be impossible for him to be present at this meeting and asking me to express his very great regret to the Association that he was unable to personally present the best wishes of the American Life Convention to our organization.

(End of business meeting.)

Dr. Fraser—We now come to the first paper to be taken up in the scientific section, and that is the paper that is to be presented by Dr. Charles R. Rein, the Assistant Dermatologist of the Post Graduate Hospital. Dr. Rein will present the paper on "The Relation of Syphilis to Life Insurance." Dr. Rein.

Dr. Rein—Mr. Chairman and Members of the Association:

THE RELATION OF SYPHILIS TO LIFE INSURANCE

The Value of the Kline Test for the Detection of Syphilis in Applicants for Insurance

By Charles Robert Rein, M. D., and Marguerite LeMoine
New York City

During the past twenty-five years, very little has been written regarding the relationship of syphilis to life insurance. There has been, moreover, a difference of opinion regarding the value of blood tests for the detection of syphilis. The performance of these tests has been cumbersome and difficult, so no insurance companies performed routine blood tests on applicants for insurance. Unquestionably, many applicants are infected but it is difficult to secure a positive history. The Metropolitan Life Insurance Company[1] was among the first to request a blood test on applicants giving a suspicious history or having lesions suggestive of syphilis. That more insurance companies have not made use of a blood test in view of the relatively high incidence of syphilis among apparently normal individuals is surprising since the statistics on the prevalence of syphilis in this country vary from 2% to 74%, dependent upon the race, locality, age, occupations, and customs of the individuals studied. The incidence in the population at large has been estimated to be about 6% to 10% of all adults.

Recently it has been shown that syphilis can be detected in a few drops of blood obtained from a bleeding socket following dental extraction.[2] During the past few months, Dr. M. H. Feldman and I have examined the blood of 3,000 dental patients taken from such a source and found 63 (2.09%) to be strongly positive for syphilis. This incidence compares very closely with that of the Life Extension Institute examinations.[3] They found that of 9,983 blood tests,

205 (2.06%) showed definitely positive blood reactions. It is most important to point out that the individuals in our series and those of the Life Extension Institute were apparently normal, healthy persons who presented themselves either for dental extractions or general health examinations.

In a recent report on the incidence of syphilis in private white patients, Kiser and Bohner[4] found that in 2,872 consecutive examinations 105 (3.65%) had a straight four plus reaction for syphilis with the Wasserman and Kahn tests. None of these patients came primarily because of known syphilis. In fact, none had a primary lesion at the time of examination and only two had secondary manifestations. At the Fite Clinic Laboratories,[5] Oklahoma, routine blood tests were performed on 2,500 white and colored individuals, of which 301 (12%) were positive for syphilis. In private colored patients, Granger[6] reported 16.5% positive blood tests.

Since syphilis is a relatively common disease, it is probable that insurance companies are unknowingly insuring a number of syphilitics and are recognizing only a few.

Dr. A. F. Hall, Jr.[7] of the Lincoln National Life Insurance Foundation recently made an excellent statistical study of policies issued by his company from 1920 to 1933, inclusive. Of the 509,672 policies issued by his company, 1890 (0.37%) were known to be syphilitic, yet according to statistics, at least 10,793 (2.09%) should have had syphilis. Of 6,768 death claims paid during this period, 118 (1.74%) were probably due to syphilis, while only 0.27% of these applicants were recognized as syphilitic at the time of issue. The company was aware of syphilis because the applicant either admitted the infection or the family physician, or other informants gave the information. The average duration of the policies among those deaths classified as syphilitic was 4.4 years, that is, the average age at issue was 42.4 years and the average death, 46.8 years. There were 46 individuals who died either of paresis, tabes, or syphilitic aortitis, and in this group, this company was aware of the presence of

syphilis in only one instance at the time the application was accepted.

The Department of Health of New York City[8] estimates that the incidence of syphilis among the people in upper New York State is about 10% and that the average life expectance for these individuals is 40 years. Certainly, if this be true, it bears out the well founded belief that syphilitics as a class, are extremely poor insurance risks.

The seriousness of syphilis not only is reflected by the increase in mortality due to the disease itself, but also in the fact that syphilis may affect seriously other medical or surgical conditions. According to the Medical Impairment Study of 1929, the mortality from organic heart disease among syphilitics is twice the normal and from Bright's disease two and one-quarter times the normal.

In addition, the syphilitic is a greater risk for surgical procedure than the non-syphilitic. Wile[9] has recently pointed out the relation of syphilis to surgery. When a patient with early syphilis (primary or secondary stages) is operated upon, the surgical wound establishes a lowered resistance which predisposes to sloughing or failure to heal, and from such areas a greater and wider dissemination takes place, followed by a greater virulence in the general infection. In latent syphilis, there may occur some sloughing, non-healing and the development of gummous syphilides in the operated field.

There are also certain syphilitic changes which mimic surgical conditions. These are among the most important phases of the surgical aspects of syphilis, as they are frequently the source of error in diagnosis and lead to surgical intervention when unnecessary. Chancres, especially the extra-genital type are frequently treated for cancer; gummous limphadenitis often simulates Hodgkin's disease, lymphosarcoma, or tuberculosis; and syphilis of the stomach is often mistaken for gastric ulcer or malignancy. Syphilis of the central nervous system sometimes presents symptons which

are mistaken for surgical conditions, such as: Gastric crises, abdominal crises, hepatic and gall-bladder pains, and uterine and bladder crises. Patients presenting these symptoms are frequently operated upon because of the similarity of their symptoms to the surgical adomen.

There are also certain surgical conditions which are caused directly by syphilitic disease, such as: Syphilitic osteomyelitis and osteitis, syphilitic strictures of the esophagus and rectum, syphilitic adhesive peritonitis, gummous arthritis, and syphilitic acute hyperthyroidism. Many of these conditions need some type of surgical intervention in addition to antiluetic therapy.

Syphilis has led to large expenditures of money for compensation. It has been known that syphilis not only delays the healing of wounds and the union of fractures, but often results in paralysis, insanity, blindness and other disabilities. In the infected individual, the syphilis not only tends to retard recovery from injury, but the injury itself may aggravate or accelerate the underlying disease. A sharp blow may be followed by the development of an extensive gumma, while slight traumatic injury to the eyes may be followed by the activation of an old syphilis with disastrous results. It can be appreciated, therefore, that syphilitics are not only bad risks because of the direct action of syphilis on the mortality rate, but also because of the indirect effect that syphilis has on other medical disorders.

The diagnosis of syphilis can be made clinically in the presence of an initial lesion, or from secondary lesions, or from the later manifestations of tertiary or cerebrospinal syphilis. But there still remains a very large group of individuals with latent or asymptomatic syphilis in whom a diagnosis is extremely difficult. It has been estimated that 50% of the patients with syphilis pass through the infective stages undiagnosed.

To attempt to secure an accurate history in connection with an insurance examination is practically useless. Many latent syphilitics never know they have, or have had, the

infection, and the majority of those who know they have, or have had, the disease, deny it for obvious reasons. Many of these syphilitics will pass the physical examination either because the infection is in its latent stages or because the examiner is not sufficiently trained to diagnose syphilis in its rarer and concealed forms. It is also well known that positive blood tests are sometimes the only definite evidence of a syphilitic infection and are not infrequently the sole means of detecting syphilis in its latent and concealed stages. A routine blood test is therefore of great importance in life insurance work, for with the single physical examination, the cursory history and the uncertain venereal history, the examiner is apt to be unaware of the presence of a syphilitic infection. A test to be of service for insurance work, must be simple in the collection of specimens, and in technic, as well as accurate.

In a previous paper before this Association,[10] we described the Kline test which can be performed with a few drops of blood easily obtained from a simple finger or ear lobe puncture. These specimens which can be collected by the field examiner, do not need the addition of chemicals to prevent clotting or bacterial contamination. They can be sent with safety over long distances to a central laboratory for examination, and are satisfactory for testing even ten days after collection.

In addition, it has been proven repeatedly that the Kline test not only is simpler in technic than the other accepted complement fixation and precipitation tests, but is more sensitive and specific.

At the present time, the test is performed on these applicants who give a history of, or present signs and symptoms that are suggestive of syphilis. A tabulated list giving these signs and symptoms is being prepared which should be of great assistance to the examiner.

The technic that has been adopted at the Metropolitan Life Insurance Company is as follows:[1]

A capillary glass pipette 5½ inches in length and 3 mm. in diameter is placed over the edge of the carrying case at an angle of 30°. The applicant's finger is then cleansed with alcohol, sponged dry and a puncture wound made with a sharp needle. The bleeding finger is squeezed and the blood is allowed to run into the capillary pipette until it is about two-thirds full. (This will yield about .2 cc. of serum which is sufficient for at least three Kline tests.) Both ends of the pipette are then stoppered with rubber policemen (a short length of small rubber tubing closed at one end) to prevent leakage and evaporation. It is replaced in the box and sent to the central laboratory for testing. Here, the rubber policemen are removed and one end of the pipette is sealed with a Bunsen burner. A fine wire is then inserted into the open end of the tube to break up the clot.

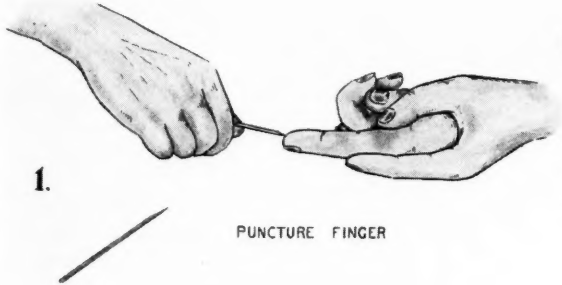
The blood in the pipette is now centrifuged for thirty minutes at high speed (about 2,000 R.P.M.) which packs the clot into the lower half, leaving the clear serum in the upper half. The pipette is then placed in a bath filled with water at a higher level than the top of the serum and kept thus at 56° Centigrade for one-half hour. After this, the pipette is filed and broken just above the clot. The serum is then allowed to run into, or is drawn into a 1 cc. pipette graduated in .01 cc.

The tests requiring the least precautions are those with the heated sera. In these, it is not necessary to determine the pH of the water and salt solutions, and furthermore, the antigen emulsions are satisfactory for use for forty-eight hours.

Materials for the Kline Tests for Syphilis

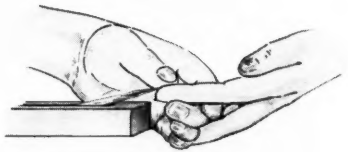
Glassware—Microscopic slides 3x2 inches as purchased, are rubbed on both sides with Bon Ami paste (prepared by breaking up a cake of Bon Ami in a small quantity of water). As soon as the paste is dry (in about five minutes), it is completely removed from the slide with a soft muslin

STEPS IN COLLECTION



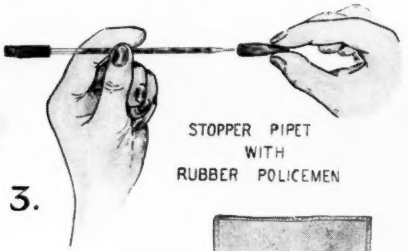
1.

PUNCTURE FINGER



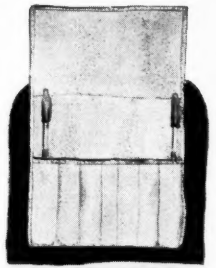
2.

WITHDRAW BLOOD



3.

STOPPER PIPET
WITH
RUBBER POLICEMEN



4.

PLACE PIPET IN JACKET
FOR MAILING

STEPS IN TECHNIQUE

1.

REMOVE RUBBER POLICEMEN FROM ENDS OF PIPET

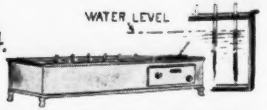
2.

SEAL TAPERED END OF PIPET IN FLAME

3.

CENTRIFUGE 30 MINUTES 2000 TO 2500 R.P.M.

4.

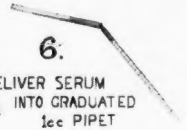


INACTIVATE IN WATER BATH 56°C. FOR 30 MINUTES

5.

MAKE FILE MARK AND BREAK AT JUNCTION OF CLOT AND SERUM

6.



DELIVER SERUM INTO GRADUATED 1cc PIPET

7.

DELIVER .05cc PATIENT'S SERUM TO SLIDE

8.

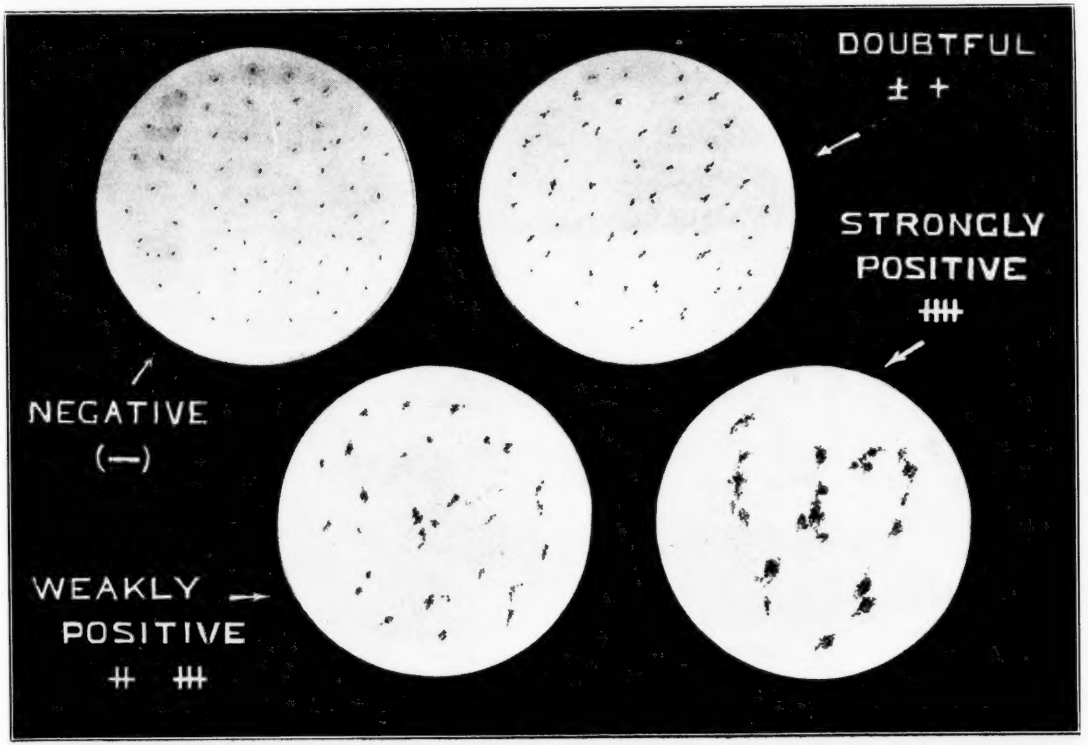
1 DROP ANTIGEN EMULSION TO EACH CHAMBER

9.

ROTATE 4 MINUTES

10.

READ THROUGH MICROSCOPE-(LOW POWER)


DOUBTFUL
± +
STRONGLY
POSITIVE
+++
NEGATIVE
(—)
WEAKLY
POSITIVE
+ ++

cloth. Upon clean slides, 12 paraffin rings, each with an inside diameter of 14 mm. are mounted. These rings are made with hot parawax by means of a thread wound wire loop, 15 mm. inside diameter.

Pipettes—The pipettes needed for delivering the sera and those for preparing the antigen emulsions are the ordinary finely graduated 0.2 to 10 cc. pipettes. The pipettes for delivering the antigen emulsions are Wright pipettes made from glass tubing 6-10 mm. diameter with the tubes about 0.5 mm. in outside diameter, for delivering a drop equal to about 0.008 cc. (62 drops per 0.5 cc.).

Slide Holders—The slide holder is a wooden lid of a slide box (3½x6¼ inches) containing an easily fitting thin wooden shelf having a small handle at each end.

*Kline Antigen**—The antigen is a lipid obtained by precipitation in acetone at 50° Centigrade to 37° Centigrade of concentrated absolute alcohol extract of beef heart muscle powder (Difco). The details of its preparation are given in a previous report.

Antigen Emulsion—The diagnostic antigen emulsion has the following formula:

0.85 cc. distilled water.

1.0 cc. of one per cent. cholesterin (C. P. Pfanstiehl) in absolute ethyl alcohol (99 plus per cent.). The cholesterin solution is prepared in about 45 minutes by placing in an oven 50°-56° Centigrade, and shaking gently a few minutes at 15 minute intervals. Kept at 37° Centigrade this solution is satisfactory for use for a few months.

0.1 cc. antigen.

2.45 cc. 0.85 per cent. sodium chloride solution.

The emulsion is prepared as follows: Into a one ounce bottle 0.85 cc. of distilled water is pipetted. The bottle is

*Standard Kline Antigen may be obtained from the La Motte Chemical Products Co. McCormich Building, Baltimore, Md.

held at an angle and the one per cent. cholesterin in absolute ethyl alcohol (pp plus per cent.) is allowed to run along the side of the neck of the bottle. The bottle is gently rotated from the neck for twenty seconds. The bottle is held at an angle again, and 0.1 cc. of antigen is pipetted against the side of the neck from a finely graduated pipette. The bottle is promptly stoppered with a cork and shaken vigorously (the fluid thrown from bottom to cork and back) for one minute.

The 0.85% sodium chloride solution is then allowed to run in quite rapidly. The bottle is again stoppered and shaken fairly vigorously for one minute.

Place the antigen emulsion in a test tube in a water bath at 35° Centigrade for fifteen minutes. The emulsion as soon as heated is ready for use.

The emulsion when examined through the microscope, at a magnification of about 120 times, shows numerous very fine particles, but no clumps whatever.

Technic

1. Place three heated serum slides each with twelve chambers on a tray in a small holder.

2. Into each of the 36 rings, pipet 0.05 cc. of the heated serum to be tested.

3. After all the sera are pipetted, one drop of the diagnostic test antigen emulsion (about 0.008 cc.) is allowed to fall from a Wright's pipette into each chamber with the serum.

4. The slides in the holder are rotated on a flat surface for four minutes.

5. The results are read at once through the microscope at a magnification of about 120 times (low power 16 mm. objective, eye piece, 12) with the light cut down as for the study of urinary sediments and reported in terms of plusses according to the degree of clumping and the size of the clumps.

Any spilling from the chamber makes the reaction therein unsatisfactory and when this occurs, the serum should be retested.

Results

During the past year, the laboratory of the Metropolitan Life Insurance Company has examined the blood of 3,230 individuals. Two or more tests for syphilis were performed on 969 specimens. On 593 of these, the Kline finger blood test and the Wasserman test were performed, while 601 had Kline and Kahn tests. The results of this comparative study demonstrated the fact that the Kline test was more sensitive and specific than the Wasserman and Kahn tests performed on blood obtained by vena puncture.

In 2,250 individuals, only the Kline finger blood test was performed. In this series 177 (7.86%) gave definitely positive reactions (two, three, or four plus). This percentage is somewhat higher than one would normally expect and probably is due to the fact that the Kline test was performed on those individuals who presented symptoms or gave histories which were suggestive of a syphilitic infection.

Several other companies are now performing the Kline test on applicants for insurance.

Comment

In addition to the diagnostic Kline test described in this paper, we are also able to perform the test with a more sensitive antigen called the exclusion emulsion Kline test which is so sensitive that when the test is negative it excludes syphilis in that particular individual. The Kline exclusion test may also be used as a form of syphilimetry in syphilitic patients who are under treatment. Evidence is accumulating to show that when the blood of an adequately treated syphilitic gives a negative exclusion test on three successive occasions, and the exclusion test on the spinal fluid is also negative, that individual is serologically cured of his disease.

Dr. Leo Spiegle (Clinical Professor of Dermatology at Bellevue Medical School) and I have made a clinical and serologic study of 871 tests on 414 individuals under treatment and observation in the various stages and types of syphilis at the Lenox Hill Hospital. We have quite a number of patients who have received adequate standard treatment and are free from all clinical evidence of syphilis, who also have negative exclusion Kline tests with their blood and spinal fluids. These patients are then clinically and serologically cured.

A similar study is being carried out by Dr. Kline and his associates at the Mount Sinai Hospital in Cleveland. They have found that in a follow-up study of several years that the exclusion test is a thoroughly reliable criterion, not only of a serologic, but of a clinical cure.

This has a very important bearing on insurance work because it offers more definite criteria for the determination of a cure from syphilis.

Conclusions

1. The mortality among syphilitics is high and yet the disease is widespread. Thus it is evident that the insurance companies who accept syphilitic applicants will experience greater losses than those companies whose applicants are free from syphilis, or whose syphilis is influenced by judicious treatments.

2. We have described a simple precipitation test for the detection of syphilis in suspected cases. In addition, we have discussed the more sensitive exclusion test which may be used as a criterion for the determination of a serologic cure.

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DR. FRASER—The discussion of this paper will be carried on by Dr. Ralph G. Stillman of New York City. Dr. Stillman.

DR. STILLMAN—I shall have very little to say this morning in respect to the relation of syphilis to life insurance, since I know comparatively little about the life insurance aspect of the problem. My sole contact with a similar situation occurred some years ago when the laboratory of the New York Hospital carried out blood sugar examinations for one of your companies for a period of some two years. During that time I was impressed with the reluctance, for the most part, of the agent to have the examination made, while as a rule the applicant was much interested. I imagine that since that time educational work has brought about a lessening of the difficulties in obtaining consent to a venepuncture.

There is certainly no question as to the importance of syphilis in influencing life insurance risks and the frequency of the disease is far greater than is appreciated by most men who are not familiar with figures such as Dr. Rein has presented this

morning. Since we serve a hospital population, we do not have wholly comparable figures. However, of 7,095 specimens from the Woman's Clinic, 231 or 3.2% gave a four plus Kline reaction. Since the majority of these patients are on the obstetrical service, this figure is the nearest approach we have to the situation in normal individuals.

I can, however, tell you something of the experience of a general hospital with the Kline test. The practice which we have adopted at the New York Hospital is as follows:

All specimens of blood that are sent in for a serologic test for syphilis are subjected to the Kline test. If this test is negative, then no further examination is made, unless the patient happens to have had syphilis or unless the case is unusually obscure and the clinicians are anxious to have the examination complete. In such instances, the sera are saved until the following day when the Wassermann tests are carried out. If the Kline test is positive in any degree, then the Wassermann test is performed. This practice, as you will see from the figures, has been tremendously saving in the reduction of the number of Wassermann tests performed in our laboratory. In the two years ending August 31, 1934, we carried out 25,444 Kline tests and 8,519 Wassermann tests. Thus you will see that on 67.3% of the specimens only the Kline test was done and we were able to omit the Wassermann in 17,125. This represents a large saving not only in time, but in money. During this period the cost of the antigen has been in the neighborhood of 0.6 cent per test.

We have been carrying out the Kline test now for about 5 years and during that time many of the clinicians have been frightened at the relatively high percentage of positive Kline tests not infrequently associated with the absence of any positiveness in the Wassermann reaction. In our series of 25,444 Kline tests derived from all parts of the Hospital and including the Out-Patient Department, 2,448 or 9.6% yielded a strongly positive (++++) reaction. Of the 8,319 Wassermanns, however, only 839 or 10.0% were four plus. I haven't the figures for

the reactions of a less degree of positiveness, but you will note that there were nearly three (2.91 exactly) four plus Klines for each four plus Wassermann. Since many of these patients are not obviously syphilitic, there have been innumerable discussions as to the desirability of continuing a test that is so sensitive. It is my practice to say to these men that under no circumstances is it justifiable to base a diagnosis of syphilis upon a single positive serologic reaction of any kind in the absence of supporting historical or clinical evidence. At the same time I point out that the complete exclusion of such evidence is a matter of great difficulty since a physical examination directed toward this end must be far more complete than that usually given to a patient even in the better hospitals. However, if the Kline test is repeatedly strongly positive then no matter what the result with the Wassermann or any other test, serious consideration must be given to the possibility that the patient is suffering with syphilis even though it may be for the time being entirely latent. Under such circumstances I advise the clinicians that the patient should be followed up at intervals so that any return of the activity of the disease may be immediately detected or he should receive anti-syphilitic treatment in an attempt to prevent the appearance of active disease and to determine the influence of the treatment upon the serologic reaction. This decision is, of course, not easy. It is recognized that vigorous anti-syphilitic treatment is far from innocuous but the satisfaction of preventing a return of the activity of the disease probably justifies the institution of treatment. If the Kline test becomes negative or much weaker after treatment, then the probability of the presence of syphilitic infection is increased. We feel that the Kline test is exceedingly important in determining the need for continuation of treatment and that a patient should not be regarded as cured until the Kline test is completely negative.

So far as the technic is concerned, we have uniformly carried out the Kline test on inactivated serum practically as described by Dr. Rein and it is our impression that this technic is simpler than that of other flocculation tests. The tests at the New York

Hospital have been carried out over a period of years by five or six different technicians at different times and in every instance they have been able to develop within a comparatively brief period, a rapidity and accuracy of technic which we have never been able to develop with some of the other flocculation tests. Furthermore, the Kline antigen seems to be more nearly standardized and the result is easier to read than in some of the other tests. We have had no experience with the test on defibrinated uninactivated blood but it should prove very helpful under certain circumstances.

It seems to me on a broad basis that the more reliable tests an insurance company carries out upon its applicants, the more accurate will be its selection of risks and if it is possible by the introduction of a procedure such as the Kline test to recognize a fairly large percentage of applicants who should be classified as inferior risks, the companies would then be able to figure their costs and assess their premiums more accurately. This would result in benefit to the entire population.

I think it is proper here to express our appreciation of the assistance which Dr. Rein has given in connection with the introduction of the Klein test in this general vicinity and especially in the New York Hospital. I thank you.

Dr. Fraser—Is there any member present who would like to take further part in this discussion or has any remarks to make in regard to this test? If not, I will ask Dr. Rein if he wishes to make any further remarks in regard to the discussion of his paper.

Dr. Rein—I would just like to add one more word regarding the test by other groups besides insurance groups. Various hospitals have now adopted the defibrinated finger blood test. A few drops of blood are taken and defibrinated. This is especially important in connection with donors for transfusion. In New York City there is a Transfusion Board that examines the donors once a month, which means that if a professional

donor is examined today, he is available and is used for thirty days. Unfortunately we have seen a number of individuals who contracted their syphilis and a couple of days later became markedly infected and developed chancers almost immediately and showing four plus reaction by the time the transfusion was to begin. A number of cases of syphilis have been transmitted by syphilitic donors.

A large number of hospitals have family donors. When a patient needs a transfusion, all the members of the family come down and have their blood typed. Blood tests are not done on these individuals and it has been found that a large number have syphilitic blood.

I personally feel that all four plus donors do not give the disease to the recipient, but there is an article in this month's issue of the Urological and Cutaneous Review by a dermatologist over in Russia who reported a large number of cases transmitted by latent syphilitics.

I believe the Transfusion Board is inadequate to rule out syphilis in donors if they are examined only once a month and the nurse or doctor or interne does not take time out to give the donor a very thorough examination for syphilis. A blood test should be done at the time of transfusion. It takes only four or five minutes for a finger blood test and syphilis can then be ruled out in every single individual, even those with four plus reactions. It certainly is an unnecessary danger to use a syphilitic as a donor when all hospitals are in a position to make use of this defibrinated finger blood test.

I thank you.

Dr. Fraser—No doubt you have, as we have, during the past year received considerable criticism from various Chambers of Commerce, Health Societies and Health Boards because of the fact that we have called for statements from various organizations that have been carrying out periodic examinations. To meet that criticism, I asked one of our new members—in fact, one of our baby members—Dr. Linford H. Lee, of the Pacific

Mutual Life Insurance Company, to give us a report on a study of health examinations that has been made by the Pacific Mutual. I will now call on Dr. Lee.

Dr. Lee—Mr. President and Members of the Association: Right now I am a typical example of one of those emotional blood cases you gentlemen will not take.

A STUDY RELATING TO HEALTH EXAMINATIONS

By L. H. Lee, M. D.
Assistant Medical Director
Pacific Mutual Life Insurance Company

Dr. Fraser, President of the Association, has asked me to present at this meeting the results of a study made by the Medical Department of our Company relating to health examinations.

In making the study we were not trying to build argument for or against the general subject of health examinations. We were interested only in trying to find the correct answer to this question—"Are we justified in accepting without investigation a personal history of health examinations when given by an applicant for insurance?" This history is obtained in the answer to the question in our medical form—"What physicians have you consulted in the last seven years?" For our purpose we grouped all cases under one heading where there was a history of health examinations whether it was described as such or as a routine, periodic or general examination. In practically all instances a history of symptoms was not admitted and it was stated that the results were negative. You are familiar with the explanations which are frequently given. The most common reason is that the applicant accompanied some relative who was ill when he went to the doctor, hospital or clinic, and while there thought it a good idea or a good way to pass the time and had an examination made. Another common reason is that the applicant's wife thought he was working too hard and insisted that he go to the doctor to be sure he was all right. Sometimes the doctor would be partner in a scheme to frighten him so that he would take better care of himself. Other equally interesting explanations are given.

Previous to 1930, a history of health examinations was not always investigated by our Company beyond the examiner. Since that time it has been our practice to ask for an attending physi-

cian's statement in all such cases. A special form letter is used requesting not only the attending physician's findings but particularly what symptoms prompted the examination. In his reply to our request, the attending physician will sometimes criticize our action and state that we are not in step with medical progress, and that he or the group with which he is associated advocates health examinations and periodical check-ups for all patients. He will further state that in asking for an explanation, insurance companies are impeding the progress of those who are trying to educate the public relative to the importance of such a procedure, and that our attitude is discouraging. Our answer to the criticism is that we recognize the value of health examinations but we do not believe that those who are interested in promoting the idea have succeeded in popularizing it and that the majority of individuals who do have such examinations either have knowledge of an impairment or have symptoms of organic disease or functional disturbance.

Experience in medical practice has been that it is most difficult to persuade healthy individuals to submit to examination unless they are connected with an institution where it is compulsory. Furthermore, it is only those who have findings or symptoms who are inclined to return for repeated check-ups. Even the average impaired individual with a stable nervous system, who is actually at work, is not inclined to have periodical examinations. The average healthy individual, who is not neurotic, is not much concerned about his physical condition, and while he may admit that he should be checked-up he will do so only under pressure, and if it does not interfere with his activities. Therefore, we made this study in an effort to determine whether actual experience would substantiate our opinion.

We studied 20,000 applications for insurance. The applicants were of the male sex, ages 21 to 50, in high grade business and professional occupations with average earning capacity of over $3,000 per year; geographically widely distributed, coming from practically every section of the country. We limited our study to this type of applicant believing that an educational program promoting the idea of health examinations would be much more

apt to reach and appeal to this type of individual because of his responsibilities and economic value. He should be desirous of knowing his condition and following the advice of the doctor as to the maintenance of health and efficiency. We selected for our study only those in the above classification.

Of these 20,000 cases—291 gave a history of health examinations with negative results. This is approximately 1½%. This result we believe is evidence to support our contention that the idea is not popular in the group most susceptible, and that a history of health examinations justifies suspicion. We secured statements in 244 of these 291 cases. A study of 47 cases where statements were not received is interesting. Information received in six of them within two or three years after issue revealed the following histories:

2 Gall bladder operations. Investigation revealed a chronic history.
1 History chronic sinusitis and chronic cough.
1 History weight loss and unhealthy appearance.
1 History chronic amoebic dysentery.
1 Disability claim (no details).

This shows 13% of this group were not good risks. It was because of this type of experience that we made a general rule requiring attending physicians' statements in all cases where history of health examination is given.

Of the 244 cases where statements from attending physicians were received, the following facts were revealed:

Only 33 had no history and the physical examination was purely a matter of check-up. This is approximately 13%.

All of the others—211 out of 244, were prompted to have an examination because of some existing impairment or symptoms. I will not attempt to give details concerning each case although each record is most interesting. These 211 individuals had an examination for the following reasons:

41 History of gastrointestinal disease or symptoms relating to the gastrointestinal tract.

29 History of nervous disease or symptoms—most common condition, chronic nervous exhaustion and fatigue states.
28 Existing heart lesions or symptoms relating to the heart.
19 History of abnormal urinalysis; glycosuria; albuminuria or hematuria.
13 History of weight loss or underweight.
9 History of disease of the genitourinary tract or symptoms relating thereto.
8 History of hypertension.
7 History of some rheumatic condition.
7 History of positive or suspected tuberculosis or asthma.
6 History of recurrent or chronic upper-respiratory condition.
4 History of thyroid trouble.
4 History of visual impairment.
4 Poor family history—tuberculosis or circulatory.
3 History of syphilis.

The remainder, or 28 cases, had complaint of many other conditions and have been classified under miscellaneous.

This information clearly indicates that only a small proportion of individuals who have health examinations are free of impairments or symptoms.

I want to admit that this study may not present the correct picture in that it was limited to applicants for insurance. It is probable that many who have no impairments would not apply and that there would be a strong tendency for those who were impaired to seek protection.

Nevertheless, these figures are interesting and in our opinion indicate the answer to our original question—that a history of a health examination when given by an applicant for insurance cannot be disregarded without proper investigation.

DR. FRASER—We did not ask for a discusser to cover the remarks made by Dr. Lee, but if there is anyone in the meeting who would like to take part in the discussion, I should be very much pleased to have him come up. In case no one

has anything to say upon the matter, I will call on Dr. Whitney to make a few remarks regarding the luncheon and report for the Dinner Committee.

Dr. Whitney—Those who have not registered at the desk and are staying for luncheon today will please register immediately after the meeting. The dinner tonight will be at the Manhattan Club, 26th Street and Madison Avenue, at 7:00 o'clock sharp. Those who have not registered for the dinner tonight will kindly do so at the desk. You may also register for tomorrow's luncheon at the same time. Luncheon will be served in the Astor Gallery adjoining this room at 1:00 o'clock sharp.

Dr. Fraser—This will conclude the morning meeting. So we will now have a recess until luncheon at 1:00 o'clock.

Dr. Fraser—The next subject on the program is the paper contributed by Dr. Dublin, Dr. Jimenis and Mr. Marks of the Metropolitan Life Insurance Company, and the discussers will be Dr. Russell S. Fowler of Brooklyn and Dr. Burrill B. Crohn of New York City. Dr. Dublin will open the proceedings by giving a short résumé of his paper and Dr. Jimenis will finish the discussion. Dr. Dublin.

FACTORS IN THE SELECTION OF RISKS WITH A HISTORY OF GALL BLADDER DISEASE

By Louis I. Dublin, Ph. D., Third Vice President and Statistician, Albert O. Jimenis, M. D., Assistant Medical Director, and Herbert H. Marks,

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I

Gall bladder disease is a very common ailment in adult life. Blackford[1] reported the condition in 6 per cent. of his patients over 20 years of age in a large Western clinic. Crump[2] and Mentzer[3] found the incidence of gall bladder pathology on routine autopsy ten times as high. Nor was this high incidence confined to the late adult ages. Mentzer reported gall bladder pathology present on autopsy in one-third of all cases in the twenties and in over two-thirds at every period of life over 30. Crump reported about a two-fifths incidence on autopsy in the twenties and thirties and thereafter a gradually rising percentage. Stones were present in approximately one-fifth to one-half of these cases, the higher figures being reported at the older ages. In the series reported by Mentzer, only 15 per cent. of those showing pathological conditions in the gall bladder had given clinical evidence of gall bladder disease. Obviously a relatively small fraction of persons in apparent good health are aware of the existence of this particular impairment. I have in mind especially the opinion of Dr. Draper, George Draper of New York, according to whom those who have gall bladder disease conform to a very definite constitutional type. He calls them the lateral as contrasted with the linear type. Those who have gastric ulcers,

1. Blackford, J. M., King, R. L., and Sherwood, K. K.: Journal of the American Medical Association, Vol. 101, page 910, (September 16) 1933.
2. Crump, C.: Surgery, Gynecology and Obstetrics, Vol. 53, page 447, (October) 1931.
3. Mentzer, S. H.: Surgery, Gynecology and Obstetrics, Vol. 42, page 782, (June) 1926.

for example, would correspond to the linear type—tall and thin—in contrast with those who have gall bladder disease and who are short people, heavy set, usually with a broad, barrel trunk and over-weight. That is the picture that Draper has in mind. More women than men conform to this type. Draper went so far as to say that he could go down the aisle of a morgue in a hospital and pick his gall bladder cases. Well, there are those who question his skill in that regard, and my only purpose in bringing this point up at all is to fix the idea that these gall bladder cases do, after all, in many, many cases correspond to a certain type of build, and I think that is an important point for us to keep in mind.

It is not surprising, therefore, that a history of gall bladder disease is often given by applicants for insurance. Next to appendicitis it is the most frequent serious surgical history. This explains the interest of life insurance medical directors in the prognosis of the condition as is manifested by the number of previous studies.

In the Specialized Mortality Investigation of 1903, applicants reporting hepatic colic or gallstones were studied as a group, and it was then discovered that their mortality was somewhat above normal, particularly for applicants in their mid-forties or early fifties. The subject was again studied in the Medico-Actuarial Investigation of 1912. Two groups were distinguished, "gallstones and hepatic colic" and "bilious colic". The mortality of both groups reporting a single attack was nearly equal, about 130 per cent. of the Medico-Actuarial Mortality Table. The former group was analyzed according to the time elapsed since illness, but all classes showed approximately the same excess mortality. Applicants reporting more than one attack, however, suffered a mortality nearly twice the expected.

The experience on gall bladder cases was again investigated in the recent Medical Impairment Study—1929. In this instance, attention was paid more to the type of treatment than to the condition for which the gall bladder was treated. It was found that, on the whole, the lowest mortality prevailed among those

reporting removal of the gall bladder. In the standard cases, the ratio was 98 per cent. of the Basic Table. Medical cases showed the next best mortality and drainage cases the worst. Only the medical cases were studied according to time elapsed. Ratios of 120 per cent. of the Basic Table were recorded on standard cases insured at any time within five years of illness, but curiously enough similar substandard cases had a mortality only 83 per cent. of the expected and actually lower than standard cases insured from six to ten years after illness. Medical cases with recurrent attacks showed a mortality about one-fifth higher than normal and there was practically no difference between the standard and substandard groups. We will find similar discrepancies in our studies. It seems that in this condition we are confronted with all kinds of cross currents of findings. There isn't a consistent picture, which I think arises from the fact that we have insufficient data.

II

Ours does not pretend to be a final study of the subject. Limited as it is to the experience of a single company, the number of cases is not large enough to permit thorough investigation of the various factors involved. While certain of our results are definite and valuable, others are somewhat confusing. Consequently, our study leaves many important questions unanswered. We, therefore, present our results to you as an additional investigation of the subject, but with a view to enlisting interest in a more extensive research, which is possible only through the cooperation of several companies.

Certain limitations regarding the diagnosis of gall bladder disease should be pointed out at the beginning. We all know how difficult it is in these cases for the physician to make an accurate diagnosis. The number of conditions which cause symptoms in the upper right quadrant of the abdomen are legion, and in a study, such as we have made, we must depend largely upon the statement of the applicant, especially since we do not usually refer for details to the physician who treated the applicant. This

is partly because of our liberal attitude towards such applicants, as well as for administrative reasons. Diagnosis is, of course, more certain in operative than in medical cases.

The present paper relates to the experience of the Metropolitan Life Insurance Company with 2,720 white applicants accepted for insurance during the years 1912 to 1928 and traced to their policy anniversary in 1933. All of them were free from other medical impairments or histories serious enough to cause the applicant to be refused standard insurance. They were, likewise, employed in standard occupations. We shall report also on a small identical group, but who in addition to their gall bladder disease were substandard risks because of overweight alone.

Before discussing our mortality results, a brief outline of the main characteristics of the group will be in order. The disproportionate frequency of gall bladder disease among women is clearly shown by their number, 1,372, which is slightly in excess of the number of men, 1,348. In our Ordinary Department in the corresponding period, female applicants constituted only 15 per cent. of the total. These figures indicate that the condition is five to six times as common in women as in men and confirm clinical experience. Of the 2,720 applicants, 463 were under age 30 at issue; 1,034 between ages 30 and 40; 837, between 40 and 50; and 386, 50 or over. Thus, slightly more than half were less than 40 years of age. Taking into account also the time elapsed between illness and application for insurance, the number having the disease in early adult life was considerable. Women reporting the condition were, on the average, somewhat younger than men. Thus, 20.8 per cent. of the women were under 30 and 62.6 per cent. under 40, compared with 13.1 per cent. and 47.3 per cent. respectively for men. The vast majority of these cases were insured within ten years of their illness, the average interval being between three and four years. As for the build of these applicants, a very different picture existed as between men and women. Among men, overweight was probably only a little more frequent than usual, but in women it was very common. Thus, 51.8 per cent. of the women were at least 5 per cent. above average weight, and 22.8 per cent. were at least 15 per cent.

above average, compared with 39.9 per cent. and 11.9 per cent. respectively for men. These overweights were all within the limits of standard insurance.

We have classified these applicants according to the type of history, namely, whether stones were present or not, whether operated upon or treated medically†; and for the operated group, whether the gall bladder was excised or simply drained. These are, of course, not mutually exclusive groupings, but they do bring into relief the questions usually associated with the degree of severity of the disease and the prognosis.

As Table 1 shows, 55 per cent. of these applicants gave a history of gallstones; only 15 per cent. of simple inflammation; and the remaining 30 per cent., no details as to the nature of the trouble. Over 80 per cent. of the cases reported operative intervention and in this group removal of the gall bladder was the operation specified most frequently, namely, 999 cases, compared with only 300 reporting drainage. The number in which the type of operation was not specified (974 cases), was, however, almost equal to the number specifying removal. This detail regarding the operation was most often absent in the case of gallstones and, in fact, these cases were most numerous (833). It is interesting to note that the second largest group (514 cases) consisted of those reporting removal of the gall bladder without further details. Medical treatment was specified relatively most often in applicants with a history of inflammation.

About 500 of these 2,720 applicants gave a history of two or more attacks or of illness from the disease lasting at least three months, and in half of these the history of the condition extended over a period of a year or more. In over one-fifth (498) of the operated cases, the appendix was also removed.

Certain other characteristics of the group are of interest. The proportion with surgical treatment, especially removal, was much higher in women than in men. In the latter, drainage and medical treatment were more common. Women reported a history of stones with greater frequency than men. Applicants with a history of stones tended to be older than the others.

†The medical group includes those not specifying operation.

TABLE 1.

Reason and Type of Treatment and Severity of Condition Reported by 2,720 Applicants with a History of Gall Bladder Disease. Metropolitan Life Insurance Company, 1912-1928.

Reason for Treatment; Severity of Condition	Total Cases	Type of Treatment			
		Operative			
		Removal	Drainage	Not Specified	Medical
All Cases	2,720	999	300	974	447
A. Reason for Treatment					
Gallstones	1,480	364	103	833	180
Inflammation of gall bladder	400	121	65	39	175
Not specified	840	514	132	102	92
B. Severity of Condition					
Single attack, or illness of short duration, uncomplicated	1,718	538	191	660	329
Recurrent attacks and/or illness lasting 3 months or more	504	207	48	131	118
Chronic disease (duration, 1 year or more)	254	112	24	58	60
Operated cases* reporting simultaneous appendectomies	498	254	61	183	—

*Excludes chronic or recurrent cases.

Of the entire group, 70.6 per cent. were accepted for standard insurance; 23.7 per cent. were limited to our Intermediate plan and only 5.7 per cent. to our Special Class plans. In general, we were more liberal where the gall bladder was removed and more severe in cases treated medically than in those reporting drainage. Applicants reporting treatment for stones by drainage or non-surgical methods were rated more frequently than other

cases treated by these methods. But generally speaking, we are dealing with a group insured at standard rates.

III

We have studied the mortality of these cases in relation to the several factors which might be expected to affect the mortality, namely, the sex and age of the applicant, the type of treatment, the presence or absence of stones, build and the interval since the attack. We have combined standard and substandard cases on the assumption that they would tend to separate out in the analysis of the several factors involved. We do, however, give the aggregate results on the standard and substandard issues separately.

The aggregate results are presented in the form of ratios of actual to expected deaths by the American Men Mortality Table. Ratios have been computed first on a select and ultimate, and, second, on an ultimate basis, but we shall quote the ratios by the first of these. The other ratios will be found in the tables.

Mortality—All Cases

The 2,720 risks with a history of gall bladder disease yielded a total of 17,486 years of life, an average of 6.4 years. There were 159 deaths, or 105.7 per cent. of the expected by the American Men Table. The mortality was a shade better in the sixth and later years after issue than in the earlier insurance years; a little better over age 40 than under 40.

Mortality in Relation to Severity and Simultaneous Appendectomy

The first step in our analysis was to assess the hazard of the reported severity of the disease, and the extra risk, if any, in those applicants with operative histories who had the appendix removed at the same time. As Table 2 shows, applicants whose history, as given, indicated but a single attack, made up the bulk of this experience. They showed no important departure from the average for all cases. Applicants who reported either recur-

rent attacks or illness running over a period of three months or more, likewise had a mortality not much different from the group as a whole, and this was also true even for those who

TABLE 2.

Mortality of Risks with a History of Gall Bladder Disease in Relation to Reported Severity.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Broad Attained Age Groups.

Attained Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Total, all cases	17,485.67	159	105.7	94.6	108.7	102.3
Under 40	6,699.55	32	114.3	100.6	106.2	137.4
40 and over	10,786.12	127	103.7	93.3	109.6	98.3
Single attack or short illness, uncomplicated	10,987.97	103	105.6	94.5	102.4	109.3
Under 40	3,976.44	20	120.6	106.2	105.5	164.3
40 and over	7,011.53	83	102.6	92.0	101.4	103.7
Recurrent attack and/or illness lasting 3 months or more	3,393.74	35	112.8	101.9	149.0	80.0
Under 40	1,267.08	8	150.9	132.5	156.3	**
40 and over	2,126.66	27	104.9	95.3	146.4	74.3
Chronic disease (duration 1 year or more)	1,710.83	17	107.1	97.1	161.1	59.3
Under 40	594.74	3	**	**	**	**
40 and over	1,116.09	14	104.6	95.5	178.6	**
Operated cases with simultaneous appendectomy*	3,103.96	21	95.7	85.4	87.3	107.0
Under 40	1,456.03	4	**	**	**	**
40 and over	1,647.93	17	107.4	96.3	99.5	115.5

*Exclusive of those reporting recurrent or chronic attacks.
**Ratio not significant because of small number of deaths.

definitely reported the condition as lasting for a period of at least one year. In these cases there was, however, a distinctly high mortality in the early years after issue, but the subsequent experience, both under and over age 40, has been excellent. The results indicate that in these cases we simply obtained fuller details, and that they were not essentially different from the other cases. In like manner, the experience on those who reported a simultaneous appendectomy differed little from the group as a whole. In fact, it was slightly better. This result need not surprise us because these risks must have been in excellent condition to warrant the double operation, and we do not know whether the appendicitis or gall bladder disease was primary.

In view of the slight differences in mortality between these cases specifying recurrent or chronic gall bladder trouble and those reporting simultaneous appendectomies, on the one hand, and all other cases, on the other, we shall disregard these distinctions in the rest of this paper.

Mortality According to Age—All Cases

In the group, as a whole, the mortality ratios varied surprisingly little according to *attained* age, except at ages 65 and over. (See Table 3.) There, the mortality was 142.6 per cent. of the expected. There were differences according to attained age between the mortality in the first five and later insurance years, but where we had a sufficient number of cases and deaths the tendency was for a higher mortality in the early years after issue, or for only a slight difference between the two periods.

Similar results were obtained by *ages at issue.* (See Table 3.) In the aggregate, relatively small differences were recorded for those under 50 years of age at issue, but for those over 50 the mortality was appreciably worse than for the rest. The experience on these older risks was marked by an extremely high mortality in the first five years after issue, namely, 141.1 per cent. of the expected. The ultimate experience at older ages was, however, about the same as for the younger risks.

TABLE 3.

Mortality of Applicants with a History of Gall Bladder Disease According to Attained Age and Age at Issue.
Ratio of Actual to Expected Deaths by the American Men Mortality Table.

Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
All Ages	17,485.67	159	105.7	94.6	108.7	102.3
Attained Ages						
Under 25	340.02	....	....	....	....	....
25-34	3,281.19	14	108.3	95.6	89.1	176.7
35-44	6,559.29	36	105.7	93.4	117.9	82.9
45-54	5,179.12	55	107.9	96.3	104.0	112.2
55-64	1,921.30	40	96.2	86.8	123.4	75.8
65 and over	204.75	14	142.6	136.7	*	155.6
Ages at Issue						
Under 30	2,717.05	11	98.7	90.1	73.7	137.6
30-39	6,649.84	38	108.5	98.1	114.0	101.7
40-49	5,674.81	49	92.2	82.0	81.1	102.8
50 and over	2,443.97	61	120.3	104.4	141.1	96.8

*Ratio not significant because of small number of deaths.

IV

Mortality in Relation to Sex

A very striking feature of this experience was the distinct difference in mortality of men and women with a history of gall bladder disease. The women showed up exceedingly well. As Table 4 shows, there were only 55 deaths among them, or 80.6 per cent. of the expected, compared with 126.6 per cent. among the men, based on 104 deaths. Among females, the mortality was appreciably higher in the first five years after issue than in the later years, whereas among men the mortality was practically the same in both periods. In both sexes, the mortality was somewhat higher at ages under 40 than at ages 40 and over. A distinguishing feature among males was the high mortality, nearly twice the expected, in the small ultimate experience at ages under 40.

TABLE 4.

Sex Differences in the Mortality of Risks with a History of Gall Bladder Disease.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Type of Treatment and by Broad Attained Age Groups.

Sex; Type of Treatment; Attained Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Males						
All Cases	8,901.75	104	126.6	113.4	126.0	127.2
Under 40	2,788.86	16	137.8	120.5	116.4	198.7
40 and over	6,112.89	88	124.8	112.2	128.5	121.4
Removal	2,845.21	25	102.7	91.1	127.0	68.8
Under 40	934.60	5	129.5	112.1	*	*
40 and over	1,910.61	20	97.6	87.0	125.7	64.2
Drainage	1,070.56	21	233.3[a]	208.1	282.0	182.2
Under 40	340.73	4	*	*	*	*
40 and over	729.83	17	223.7	200.7	278.6	174.6
Operation—type not specified	3,080.46	35	117.9	105.7	97.6	136.9
Under 40	843.70	6	167.6	148.1	*	*
40 and over	2,236.76	29	111.1	99.8	93.5	125.5
Medical	1,905.52	23	120.4	109.1	89.6	147.3
Under 40	669.83	1	*	*	*	*
40 and over	1,235.69	22	134.6	122.7	114.8	149.4
Females						
All Cases	8,583.89	55	80.6	72.2	89.6	70.1
Under 40	3,910.70	16	98.3	86.3	99.9	*
40 and over	4,673.19	39	75.1	67.6	84.5	66.4
Removal	3,035.78	19	88.4	77.9	88.8	87.6
Under 40	1,489.75	7	113.8	99.2	123.7	*
40 and over	1,546.03	12	78.2	69.3	69.3	89.7
Drainage	852.69	5	65.6[a]	58.5	*	*
Operation—type not specified	3,470.27	21	70.9	64.1	102.2	40.2
Under 40	1,432.18	6	99.5	87.8	*	*
40 and over	2,038.09	15	63.6	57.8	106.4	*
Medical	1,225.15	10	105.3	95.1	*	124.0
Under 40	607.41	1	*	*	*	*
40 and over	617.74	9	129.3	117.2	*	151.5

*Ratio not significant because of small number of deaths.

([3]Dr. Dublin emphasized the fact that the mortality on drainage cases among men was 233.3% of the expected but the experience on women was favorable, 65.6% of the expected.) That looks on its face as rather absurd, and yet there is a goodly experience here and I have no way of explaining this tremendous difference as between males and females. This finding of a very high mortality among males who have been drained is entirely in agreement with previous studies. However, this figure is higher than in the earlier studies. The answer is, I think, in the material, that is, in the way we have handled this material compared with the way it was handled in previous medical impairment studies. In previous medical impairment studies I suspect that the cases where operation was reported and the character of the operation was unspecified, those cases were thrown in with the drainage cases. They certainly were in our company and they may in all probability have been so treated in other companies. Now, it so turns out that the cases where the type of operation was not specified are more likely to have been cases where the gall bladder was extirpated rather than drainage cases. It appears that these cases have a lower mortality than the drainage cases, so that by the combination of the two, you get a lower mortality than you should get for drainage cases. Is that perfectly clear? In our study we were careful to keep that group out as a separate entity, that is, the cases unspecified as to type of operation, and these showed a mortality almost identical with those of the gall bladders removed. So if you are going to do any combining, you should combine them with the cases of gall bladder removed rather than the drainage cases; otherwise you will get a lower mortality for the drainage cases than you should. That makes the picture consistent. If we could get at the old material and take out these cases where the operation was unspecified as to character, I am confident we can reproduce the same disparity between drainage cases and cases of extirpation that we obtained in our study.

(Dr. Dublin then stated that medically treated cases had the next highest mortality.)

We have analyzed the experience for each sex separately, according to the type of treatment. Female risks had a better experience than men in every group so classified. Among men the mortality of those reporting drainage was especially high, 233.3 per cent. of the expected. The record was particularly bad in the first five years after issue, 282.0 per cent., but even in the subsequent years the mortality was nearly twice the standard. Males reporting medical treatment also had a relatively high mortality, 120.4 per cent. of the expected. Their mortality was notably bad in the ultimate period, namely 147.3 per cent. of the expected. The best experience among men was recorded among removal cases, namely, 102.7 per cent. of the expected. This group had a rather high mortality in the first five years after issue, but a favorable one in the ultimate period. The other males reporting surgery, but not specifying the type of operation, had a somewhat higher mortality, 117.9 per cent., than those reporting removal, but considerably less than the drainage group. In this case, the mortality was rather high in the ultimate period, 136.9 per cent. of the expected.

Among women, medically treated cases gave the worst experience, 105.3 per cent. of the expected, a result not much better than among males similarly treated. Women reporting a history of removal, who constituted half the female cases, had a mortality of 88.4 per cent. of the expected. In this group the mortality was better at ages over 40 than under. The small experience on women reporting drainage was exceptionally good, namely, 65.6 per cent. of the expected, but this result was based on only five deaths. Other surgical cases in women, where the type of operation was not specified, likewise had a favorable record, namely, 70.9 per cent. of the expected. This group was distinguished by an exceptionally favorable mortality in the ultimate years. As in the case of those reporting removal, the experience was better at ages past 40 than at the younger ages. The above differences in mortality between the two sexes as regards the details by type of treatment may, in some degree, be due to the insufficient samples at our disposal for study. We must withhold judgment regarding their significance until we assemble a greater number of observations.

V

Mortality According to Type of Treatment in Relation to Reason for Treatment

We have also analyzed our experience according to type of treatment in relation to the condition for which the applicant was treated. In this analysis we have combined the experience on the two sexes in order to have sufficiently large groups. Persons with a history of drainage gave the highest mortality, namely, 155.7 per cent. of the expected. As Table 5 shows, this unfavorable result was accounted for largely by the heavy mortality, 214.9 per cent. of the expected, among those operated upon for the relief of gallstones. The mortality was exceptionally high, nearly three times the expected, in the select period. The group drained for inflammation was too small to give significant results. The mortality was relatively high, 126.6 per cent. of the expected, in those not specifying the reason for drainage.

TABLE 5.

Mortality of Risks with a History of *Drainage* for Gall Bladder Disease. Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Reason for Drainage and by Broad Age Groups.

Reason for Drainage; Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	1,923.25	26	155.7	139.7	173.2	136.8
Attained ages Under 40	722.10	6	195.4	174.9	*	*
Attained ages 40 & Over	1,201.15	20	146.7	131.8	172.4	124.1
Ages at issue Under 40	991.85	12	247.4	226.0	209.1	303.0
Ages at issue 40 & Over	931.40	14	118.1	105.3	155.4	82.5
1. Stones	634.50	13	214.9	190.6	299.0	*
Attained ages Under 40	189.08	3	*	*	*	*
Attained ages 40 & Over	445.42	10	190.5	169.2	254.2	*
2. Inflammation	463.00	4	*	*	*	*
3. Not Specified	825.75	9	126.6	113.8	129.5	*
Attained ages Under 40	334.92	2	*	*	*	*
Attained ages 40 & Over	490.83	7	123.2	110.6	*	*

*Ratio not significant because of small number of deaths.

Those medically treated also had a higher than average mortality, 115.1 per cent. of the expected. As Table 6 shows, the mortality was highest in this group among those treated for inflammation, namely, 136.7 per cent. of the expected. The experience on those treated medically for stones was somewhat better, but still exceeded the standard, the ratio being 109.2 per cent. This result was better than that experienced on those *drained* for stones, and except at the older ages as good as those with *removal* for stones, indicating, we believe, that these medical cases were relatively mild. The experience on medically treated applicants not specifying the reason for treatment was too small to yield significant results.

TABLE 6.

Mortality of Risks with a History of *Medical Treatment for Gall Bladder Disease. Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Reason for Treatment and Broad Age Groups.**

Reason for Treatment; Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	3,131.68	33	115.1	104.2	87.5	140.4
Attained ages Under 40	1,278.25	2	**	**	**	**
Attained ages 40 & Over	1,853.43	31	133.0	120.8	109.6	150.7
Ages at issue Under 40	1,826.23	7	75.2	69.3	**	105.3
Ages at issue 40 & Over	1,305.45	26	134.3	120.6	109.3	156.7
1. Stones	1,408.06	16	109.2	99.8	83.3	127.2
Attained ages Under 40	472.42	1	**	**	**	**
Attained ages 40 & Over	935.64	15	118.7	108.9	108.0	124.8
2. Inflammation	1,110.73	13	136.7	121.8	93.3	192.8
Attained ages Under 40	463.51	1	**	**	**	**
Attained ages 40 & Over	647.22	12	158.3	141.0	**	219.8
3. Not Specified	612.89	4	**	**	**	**

*Includes all not specifying operation.

**Ratio not significant because of small number of deaths.

Applicants specifying removal of the gall bladder had a mortality of 95.9 per cent. of the expected. In this case, as Table 7 shows, there was no difference in mortality between those reporting stones and those with inflammation, the ratios being 109.1 per cent. and 106.8 per cent. respectively. The latter, however, was based upon only five deaths. Curiously enough those not giving the reason for removal of the gall bladder had the lowest mortality in this group, namely, 83.0 per cent. of the expected.

TABLE 7.

Mortality of Risks with a History of *Removal* of Gall Bladder. Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Reason for Removal and by Broad Age Groups.

Reason for Removal; Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	5,881.01	44	95.9	84.7	108.0	77.2
Attained ages Under 40	2,424.35	12	119.0	104.1	126.3	*
Attained ages 40 & Over	3,456.66	32	89.3	79.2	100.8	75.1
Ages at issue Under 40	3,248.61	15	96.6	86.8	111.7	*
Ages at issue 40 & Over	2,632.40	29	95.5	83.6	106.0	80.3
1. Stones	2,079.48	20	109.1	96.0	128.7	80.5
Attained ages Under 40	700.84	7	242.2	209.0	250.0	*
Attained ages 40 & Over	1,378.64	13	84.2	74.4	94.3	71.8
2. Inflammation	684.02	5	106.8	93.8	*	*
3. Not Specified	3,117.51	19	83.0	73.7	86.7	77.3
Attained ages Under 40	1,367.16	4	*	*	*	*
Attained ages 40 & Over	1,750.35	15	87.3	77.8	95.6	77.1

*Ratio not significant because of small number of deaths.

The mortality among the other surgical cases, in which the type of operation was not specified, was 94.6 per cent. of the expected. (See Table 8.) The vast bulk of this group consisted of those reporting a history of stones, and the cases reporting operation for relief of inflammation or not specifying the reason for opera-

tion, were so few that no comparison is possible. An important feature of this experience is the very close resemblance of the gallstone cases in this group to those specifying removal of the gall bladder, indicating an essential identity with that group. In both, the mortality ratio was higher at the younger ages, especially in the removal group. Because of the similarity of results in those reporting removal and those not specifying the type of operation, we have combined them in one of the later analyses.

TABLE 8.

Mortality of Risks with a History of *Operation, Type Not Specified,* for Gall Bladder Disease.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Reason for Operation and Broad Age Groups.

Reason for Operation; Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	6,549.73	56	94.6	85.1	100.0	89.3
Attained ages Under 40	2,274.85	12	126.4	110.6	102.2	189.4
Attained ages 40 & Over	4,274.88	44	88.5	80.1	99.3	79.8
Ages at issue Under 40	3,300.20	15	90.6	82.3	91.1	90.1
Ages at issue 40 & Over	3,249.53	41	96.1	86.2	103.9	89.1
1. Stones	5,683.78	50	93.9	84.7	106.4	82.5
Attained ages Under 40	1,871.68	10	127.7	111.7	125.4	*
Attained ages 40 & Over	3,812.10	40	88.1	79.9	101.1	78.0
2. Inflammation	270.84	3	*	*	*	*
3. Not Specified	595.11	3	*	*	*	*

*Ratio not significant because of small number of deaths.

Generally speaking, analysis of our experience according to the type of operation gave more significant results than in relation to the reasons for treatment. The aggregate results for both may be of interest and are given in Table 9. The results according to type of treatment have already been discussed separately. As to reason for treatment, those reporting inflammation of the

gall bladder, as a group, had a mortality 126.1 per cent. of the expected, or more than one-sixth higher than the ratio of 107.3 per cent. recorded for those reporting a history of stones. The mortality of those giving no details regarding their disease was lower than either of the other two groups, namely, 91.2 per cent. of the expected.

TABLE 9.

Summary by Type and Reason for Treatment of the Mortality of Risks with a History of Gall Bladder Disease.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Broad Attained Age Groups.

Type and Reason for Treatment; Attained Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Type of Treatment						
Removal	5,881.01	44	95.9	84.7	108.0	77.2
Under 40	2,424.35	12	119.0	104.1	126.3	*
40 and over	3,456.66	32	89.3	79.2	100.8	75.1
Drainage	1,923.25	26	155.7	139.7	173.2	136.8
Under 40	722.10	6	195.4	174.9	*	*
40 and over	1,201.15	20	146.7	131.8	172.4	124.1
Operation, Type Not Specified	6,549.73	56	94.6	85.1	100.0	89.3
Under 40	2,274.85	12	126.4	110.6	102.2	189.4
40 and over	4,274.88	44	88.5	80.1	99.3	79.8
Medical Treatment	3,131.68	33	115.1	104.2	87.5	140.4
Under 40	1,278.25	2	*	*	*	*
40 and over	1,853.43	31	133.0	120.8	109.6	150.7
Reason for Treatment						
Stones	9,805.82	99	107.3	96.4	121.5	93.6
Under 40	3,234.02	21	155.2	135.7	160.0	141.6
40 and over	6,571.80	78	99.0	89.4	110.6	89.7
Inflammation	2,528.59	25	126.1	112.6	96.0	167.5
Under 40	1,145.71	4	*	*	*	*
40 and over	1,382.88	21	139.8	124.8	112.8	170.5
Not Specified	5,151.26	35	91.2	81.2	89.2	93.9
Under 40	2,319.82	7	72.4	63.8	*	*
40 and over	2,831.44	28	97.5	87.1	105.4	88.6

*Ratio not significant because of small number of deaths.

TABLE 10.

Mortality of Risks Reporting a History of Gall Bladder Disease in Relation to Build*.

Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Sex and Broad Attained Age Groups.

Sex; Build; Attained Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Males						
Underweight	2,546.27	26	116.4	104.0	131.1	101.0
Under 40	901.50	3	**	**	**	**
40 and over	1,644.77	23	123.7	111.0	138.1	111.0
Normal	2,590.50	33	131.8	118.5	114.9	150.1
Under 40	794.78	5	148.4	131.9	**	**
40 and over	1,795.72	28	129.2	116.4	112.9	144.9
Overweight	3,659.00	44	130.6	117.3	130.8	130.4
Under 40	1,081.50	8	177.0	155.0	**	**
40 and over	2,577.50	36	123.4	111.3	134.3	114.1
15% or more overweight	1,132.34	14	135.1	121.5	140.0	130.6
Under 40	342.32	1	**	**	**	**
40 and over	790.02	13	144.9	131.3	151.5	139.7
Females						
Underweight	2,008.96	19	111.1	100.2	169.5	**
Under 40	920.45	6	155.0	137.9	181.8	**
40 and over	1,088.51	13	98.3	88.9	163.9	**
Normal	2,035.84	10	63.5	56.7	57.9	70.3
Under 40	915.01	3	**	**	**	**
40 and over	1,120.83	7	58.5	52.8	**	80.9
Overweight	4,443.04	25	71.4	64.2	62.9	81.5
Under 40	2,014.77	7	83.7	73.5	**	**
40 and over	2,428.27	18	67.5	61.1	62.3	72.3
15% or more overweight	1,907.13	8	59.8	53.5	67.8	**

*Classified by percentage departures from average weight for height and age as follows:
Underweight—5% or more below.
Normal —Within 5% of average.
Overweight —5% or more above, but within limits prescribed for standard insurance.

**Ratio not significant because of small number of deaths.

VI

Mortality in Relation to Build

For the group as a whole, build did not have any marked influence on the results. Because of the difference in the proportion of overweights between men and women with gall bladder disease, we studied the effect of build in the two sexes separately. The results may be of interest and are given in Table 10. Among men, underweights showed to slightly better advantage than those of heavier build. But there was no difference between those of normal weight and those who were overweight. In fact, in the overweight group, where we segregated those who were at least 15 per cent. overweight, the mortality was but very slightly higher. All weight groups showed mortalities in excess of the standard.

Among females, on the other hand, the mortality was distinctly higher among underweights than among those of heavier build. Again, there was but a slight difference between normal weights and overweights and among the latter the small group who were 15 per cent. or more overweight have thus far shown up exceptionally well.

These results are most interesting. Offhand, we should expect a distinctly higher mortality among overweights, increasing with the degree of overweight, but this was assuredly not the case. Possibly, there was some selection in favor of the overweights included in the study, in that the other groups included cases in which there had been a severe loss of weight. Certainly, this is a subject which calls for further research. We attempted analysis of the experience in these weight classes by type and reasons for treatment, but our cases were too few in number to give conclusive results.

VII

Mortality in Relation to Time Elapsed Between Illness and Application

Analysis of the mortality according to time elapsed between illness and application for insurance showed no distinct tendencies. This factor was studied separately for cases classified by type of

treatment in order to eliminate its influence, as well as for all cases combined but the number of cases was too small to subdivide further by sex. The results, shown in Table 11, were substantially negative. Among all risks insured within a year of ill-

TABLE 11.

Mortality of Applicants with a History of Gall Bladder Disease in Relation to Time Elapsed Since Illness.
Ratio of Actual to Expected Deaths by the American Men Mortality Table. By Type of Treatment.

Type of Treatment; Time Elapsed Since Illness	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
All Cases†	17,485.67	159	105.7	94.6	108.7	102.3
Less than 1 year	2,139.49	21	131.1	116.9	184.3	68.1
1-2 years	3,263.68	19	74.8	67.2	59.7	91.7
2-3 years	2,662.76	23	108.6	97.8	73.0	146.9
3-5 years	3,672.35	38	125.2	112.1	129.7	120.0
5 years or over	5,654.13	57	100.4	89.3	112.6	87.4
Operation, removal or not specified†	12,430.74	100	95.1	84.9	103.9	84.8
Less than 1 year	1,388.34	13	120.1	107.3	151.5	*
1-2 years	2,261.67	10	55.3	49.7	*	70.3
2-3 years	1,836.11	14	102.3	91.8	91.7	115.7
3-5 years	2,734.61	29	133.1	118.8	149.8	112.7
5 years or over	4,194.88	34	83.4	74.0	97.5	67.5
Drainage†	1,923.25	26	155.7	139.7	173.2	136.8
Less than 1 year	207.50	4	279.7*	246.9*	*	*
1-2 years	338.59	3	123.0*	109.5*	*	*
2-3 years	296.58	4	142.3*	129.4*	*	*
3-5 years	313.84	3	129.3*	114.9*	*	*
5 years or over	766.75	12	156.3	139.4	194.6	*
Medical treatment†	3,131.68	33	115.1	104.2	87.5	140.4
Less than 1 year	543.65	4	106.1*	94.8*	*	*
1-2 years	663.42	6	123.2	110.3	*	209.2
2-3 years	530.07	5	106.8	96.7	*	*
3-5 years	623.90	6	95.8	87.2	*	*
5 years or over	692.50	11	132.5	118.8	*	151.2

†Totals include few cases not specifying time of illness.
*Ratio not significant because of small number of deaths.

ness, the mortality was 131.1 per cent., appreciably higher than for the group as a whole. In these cases, the experience was particularly bad in the select period. The mortality in this group, insured within a year of illness, was, however, not significantly higher than for those insured three to five years after illness. Moreover, the lowest mortality, 74.8 per cent. of the expected, was recorded among those insured between one and two years after illness. Cases reporting removal or not specifying the type of operation gave substantially the same results as those recorded for all cases. In the drainage and medical groups the number of cases was too few to yield reliable ratios in detailed classes by time elapsed since illness. It may be noted, however, that in cases insured five years or later after treatment, the largest single class in both these groups, the mortality was not significantly different from the total for their respective groups.

The lack of correlation between the time elapsed since illness and the subsequent mortality is rather unexpected, and we are not prepared to state definitely why it is so. Medical selection does not explain it. If anything, the results, in our judgment, are bound up in the chronic nature of these gall bladder affections, or in the difficulties of diagnosis.

VIII

Mortality According to Rating

It is always of interest to compare the mortality results obtained with the original ratings. As we have indicated, the bulk of these cases were insured at standard rates. Table 12 shows that, in 1,919 cases included in this group, the mortality ratio based upon 98 deaths was 96.0 per cent. of the expected, compared with 121.4 per cent. based on 44 deaths in cases rated Intermediate, and 143.3 per cent. based on 17 deaths in those rated Special Class or Special Class B. On the whole, this result is relatively satisfactory, although it will be realized that in the Ordinary and Intermediate cases, the mortalities experienced were close to the limits of safety.

TABLE 12.

Mortality of Risks Reporting a History of Gall Bladder Disease in Relation to Department of Issue.

Ratio of Actual to Expected Deaths by the American Men Mortality Table.

Department of Issue	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Ordinary	12,136.37	98	96.0	85.9	101.6	89.2
Intermediate	4,107.82	44	121.4	109.0	119.3	123.6
Special Class	1,241.39	17	143.3	131.9	164.3	131.6

IX

Causes of Death

Cancer of the digestive tract and non-malignant disease of the digestive tract stood out as the principal causes of mortality in these risks with a history of gall bladder disease. Of the 159 deaths, 28 were accounted for by cancer, of which no less than 20 were of the digestive tract. Other cancer deaths were principally of the breast and genitals in females. There were also 28 deaths from non-malignant diseases of the digestive tract, in 11 of which there was recurrent gall bladder trouble. Degenerative diseases, except for a slight excess for cardiovascular disease and cerebral hemorrhage in males, accounted for a normal part of the mortality, there being 24 deaths from cardiovascular disease, 14 from cerebral hemorrhage or thrombosis and 11 from nephritis. There were 14 deaths from accidents and 4 from suicide. Influenza and pneumonia accounted for 11 deaths and tuberculosis for only 7. Because of the large number of females of more than average weight, it is surprising that there were no deaths from diabetes among them and only 3 among males. No other single cause of death was of importance.

In view of the prominence of cancer, particularly of the digestive tract, and of non-malignant digestive diseases, we have carefully analyzed the mortality from these causes in the various groups. This analysis, given in Table 13, showed several significant points. For the group, as a whole, the cancer deaths were about 50 per cent. in excess of normal, due entirely to the high mortality, twice the normal, from digestive tract cancers. The mortality from non-malignant digestive diseases was even more excessive, namely, about three times the normal.

The excessive mortality from cancer was limited wholly to males. This was twice the normal and from digestive tract cancers alone, three times the normal. Non-malignant disease of the tract in males was twice the expected. In females, the cancer mortality was practically normal, but non-malignant disease of the digestive tract rose to about four times the expected. But it should be remembered that cancer of the digestive tract is much more prevalent among males than females in the general population. A notable feature of the analysis was the relatively short duration of insurance, 3.6 years, and the low average age at death, 48.7 years, in the non-malignant digestive tract deaths, as compared with digestive tract cancers in which the average duration of insurance was 5.0 years, and the average age at death, 53.4 years.

In relation to type of treatment, we found that among those reporting removal of the gall bladder, cancer deaths were of normal frequency, while non-malignant digestive tract disease was approximately three times the normal. Among those reporting drainage, while the number of deaths involved was small, the mortality from both conditions was high, especially digestive tract cancers. Among those not specifying the type of operation, the cancer mortality was high, but the non-malignant digestive tract diseases showed approximately normal mortality. Those reporting medical treatment for their gall bladder disease showed a normal mortality from cancer, but an extremely high mortality from non-malignant disease of the digestive tract.

In relation to cause for treatment, the group reporting stones showed a mortality approximately twice the expected from cancer and a little higher from non-malignant diseases of the digestive tract. Among those reporting inflammation, while the number of cases was small, the mortality was somewhat high from both. Among those not giving the reason for treatment, cancer mortality was low, but non-malignant disease was high.

TABLE 13.

Number and Relative Frequency of Deaths Among Risks with a History of Gall Bladder Disease from All Causes, Cancer and Non-Malignant Digestive Tract Diseases. By Sex and by Type and Reason for Treatment.

Relative frequency of specified causes denoted by following symbols: =, approximately normal; +, high; ++, very high; +++, extremely high; —, low. Where no symbols appear, number of deaths was too few to be of any significance.

	All Causes		Number of Deaths			
			Cancer		Non-malignant Digestive Tract Diseases	
	Number of Deaths	Per Cent. Actual of Expected Deaths*	Total	Digestive Tract Only	Total	Recurrent Gall Bladder
Total	159	105.7	28+	20++	28+++	11+++
Males	104	126.6	17++	15+++	16++	5++
Females	55	80.6	11=	5=	12+++	6++
Type of Treatment						
Removal	44	95.9	5=	3	9++	2
Drainage	26	155.7	6++	5++	4+	2
Operation, type not specified	56	94.6	12++	9++	4	2
Medical treatment	33	115.1	5=	3	11+++	5+
Reason for Treatment						
Stones	99	107.3	21+	15++	17++	8++
Inflammation	25	126.1	6+	4+	4+	1
Not specified	35	91.2	1—	1	7++	2

*By American Men Mortality Table, Select and Ultimate Basis.

X

Experience on Risks Substandard Because of Overweight With a History of Gall Bladder Disease

Because of the very frequent association of gall bladder disease with overweight, we found as many as 476 applicants sufficiently overweight to make them ineligible for standard insurance who also had a history of gall bladder disease. These cases were in all other respects standard risks and, therefore, constituted a fairly homogeneous group. The susceptibility of fat women to gall bladder disease is evidenced by the high proportion of women, nearly 80 per cent., in this group. In respect to type and reasons for treatment, as well as in regard to the reported severity of the condition, it did not differ materially from the standard weight group to which the rest of this paper is devoted.

The experience on these stout gall bladder risks was quite favorable, a result not unexpected in the light of our analysis of build in the standard weight group. In 3,117 years of life exposed to risk on this group, there were 24 deaths, or only 94.2 per cent. of the number expected by the American Men Table. As Table 14 shows, the mortality was higher in the first five years after issue than in the later years, the mortality ratios being 112.3 per cent. and 74.3 per cent. respectively. The mortality at the younger ages was very high, nearly twice the expected, but that at the older ages has thus far been good, namely, 71.9 per cent. In this overweight group, we obtained the same favorable result for females and unfavorable result for males, as was found in the standard weight group. In the small experience on males the mortality ratio was 147.1 per cent., compared to 79.9 per cent. for females. The separate experiences by sex were too small to give reliable results according to age, but, nevertheless, there was a tendency in both sexes for high mortalities to prevail at ages under 40.

In this overweight group the number of deaths was too small to warrant detailed analysis by cause. It is noteworthy, however, that one-third of them were due to non-malignant digestive diseases, an incidence much higher than normal. There were

TABLE 14.

Mortality of Substandard Overweight Risks with a History of Gall Bladder Disease.
Ratio of Actual to Expected Deaths by the American Men Mortality Table.

Sex; Attained Age Groups	Years of Life Exposed to Risk	Deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First Five Years	Sixth and Later Years
			Select and Ultimate	Ultimate	Select	Ultimate
Both Sexes						
All Ages	3,117.15	24	94.2	84.7	112.3	74.3
Under 40	1,087.21	9	195.7	172.1	174.4	*
40 and over	2,029.94	15	71.9	64.9	90.7	54.8
Males	530.18	8	147.1	132.0	181.8	*
Females	2,586.97	16	79.9	71.8	94.3	63.7

*Ratio not significant because of small number of deaths.

only three deaths from cancer. It is interesting to learn that there were no deaths from diabetes in the group in the period under survey.

Summary and Conclusions

1. Among 2,720 risks with a history of gall bladder disease slightly more than half were females. This condition is about six times as prevalent among female applicants as among men.

2. Considering the group as a whole, the mortality was 105.7 per cent. of the expected on the basis of the American Men Mortality Table. The ratio in the select years, 108.7 per cent., was not much higher than in the ultimate period, 102.3 per cent.

3. The mortality experiences on men and women with a history of gall bladder disease were fundamentally different. These women had an approximately normal mortality, 80.6 per cent. of the expected by the American Men Table, but the men, a comparatively high mortality, 126.6 per cent. of the expected.

4. No significant differences in mortality were found between those with single or short uncomplicated attacks, those reporting

recurrent or chronic disease, and those with an operative history with simultaneous appendectomies.

5. For the group as a whole, the ratios of actual to expected deaths, considered by age, showed relatively small departures from the average for all ages combined.

6. Risks reporting drainage showed the highest mortality, 155.7 per cent. of the expected, largely due to the poor results in those drained for stones, 214.9 per cent. Among men, the drainage cases had a mortality of 233.3 per cent. of the expected, but the experience on women was favorable, 65.6 per cent. of the expected. Medically treated cases showed the next highest mortality, namely, 115.1 per cent. In this group, the results for men and women were nearly the same, the ratios being 120.4 per cent. and 105.3 per cent. of the expected, respectively. The lowest mortality ratios were recorded for those reporting removal of the gall bladder or who did not specify the type of operation. The results in these two groups were practically identical, namely, 95.9 per cent. and 94.6 per cent. respectively. Men reporting removals gave a mortality 102.7 per cent. of the expected, somewhat higher than the ratio of 88.4 per cent. recorded for women. Men with operations, the type of which was not specified, gave a somewhat higher mortality, 117.9 per cent. of the expected, than those reporting removal, but the opposite was true in women where the group with operation, type not specified, had a mortality of only 70.9 per cent. of the expected.

7. Considered by reason for treatment, the mortality was higher in those reporting inflammation, namely, 126.1 per cent. of the expected, than in those reporting stones, 107.3 per cent. Those not specifying the reason for treatment, had a mortality of only 91.2 per cent.

8. The time elapsed since illness had little effect upon the subsequent mortality.

9. Build had no appreciable or consistent effect upon the mortality. Among men the lowest mortality prevailed among underweights, but there was no appreciable difference between those of normal weight and the overweights. Among females, on the

other hand, the underweights did worse, but again women of normal weight and overweight women yielded approximately the same results.

10. Where the mortality was excessive, it was due to malignant and non-malignant disease of the digestive tract. The high cancer rate was limited entirely to males. Deaths from diabetes were of no more than normal frequency. And here I am inclined to say that as time proceeds, if we ever have a chance to study them again, I am very confident that we will find a great deal more of diabetes pictured in the mortality than we have today. Dr. Joslin, in a letter written to us after he had studied our galley remarked that there was in his judgment a great deal of diabetes in these cases, but that it was of a benign type, which might explain the rare occurrence of diabetes as a factor in mortality.

11. The experience on a group of risks sub-standard by reason of overweight, who also reported a history of gall bladder disease, was quite satisfactory. This group consisted largely of females. It would appear that this condition is fairly common among stout women, but that it carries little extra risk of mortality for them. That seems to bear out the point that Draper makes. Here is a condition that is very common among women and very common among stout women. Women who have that condition don't suffer particularly from it, subsequent mortality being rather normal, perhaps a little more than normal excepting here and there in the groups as I have tried to test them out for you by analysis.

The analysis of our experience on gall bladder risks has produced important and informative results. There is much more to be learned, of course, about such risks than can be obtained from the small group at our disposal for study. . . . Instead of having some 2,700 cases, we should have had perhaps 15,000 cases. Then we would have had something. Despite this we consider certain features very significant.

First of all this experience indicates that we should introduce a new factor into our ratings for gall bladder disease, namely, that

of sex. Why we should get such favorable results on females and relatively unfavorable results on males, especially in relation to the subsequent development of cancer, is unknown to us. But they are based on an experience extensive enough to warrant giving most female applicants with gall bladder disease a lower rating than similar male applicants.

In line with clinical experience, our analysis shows a much better mortality in the case of removal than in those with surgical drainage or medical treatment. While we have considered the type of treatment in previous ratings for gall bladder disease, we do not think it has been given its true weight. Male applicants with a history of gall bladder disease, regardless of the type of treatment are, generally speaking, substandard, or at best poor borderline risks. Even in the case of those reporting removals, who gave us the lowest mortality, there is a certain extra risk, largely of digestive disease. Most females, however, may be considered eligible for standard. Males with a history of drainage for gall bladder disease should be rated severely, particularly where there is a history of stones. The chances of recurrence or malignancy are very great. While our small experience with females in this class has been excellent, I think we are justified in being cautious with them. In regard to applicants reporting operative treatment, but without details as to type of operation, our experience indicates that we are usually dealing with a removal, particularly if stones are reported as the cause for operation. We should, however, attempt to establish definitely the type of operation in order to deal most fairly with these applicants. In these cases and in drainage cases, also, we should make more use of hospital records as the basis for rating than we have in the past.

Male applicants reporting medical treatment for gall bladder disease are, for the most part, substandard cases. Female applicants with a history of medical treatment do not warrant much lower rating than males. These medical cases, it will be realized, are a rather mixed group. Among them the reliability of the diagnosis of gall bladder disease is least satisfactory. Certainly, state-

ments regarding the presence of stones, unless confirmed at the time by thorough examination, should usually be discounted. In a sense there is probably a certain element of unconscious selection tending to segregate milder cases in this group, because most of them never develop symptoms to a degree that an operation is considered necessary.

Our experience clearly brings out the chronic nature of gall bladder disease. This, we believe, is indicated not only by the practically identical mortality of the recurrent or chronic cases, as compared with those not giving such a history, but also by the similar mortality ratios for the select and ultimate periods. It may also explain the fact that there was no appreciable variation in mortality in relation to the time elapsed between illness and application for insurance. Not much weight, therefore, should be attached to the statement of the applicant that he has had but a single attack and has been well since.

In regard to age, our results are not consistent in the two sexes nor according to the type of treatment. There may be good reasons for this, but we must defer judgment on this matter until we get a larger experience. In like manner, our results according to build are different for the two sexes and for the various groupings by type of treatment. Again, there may be very sound reasons for this, particularly in regard to the relatively favorable results on overweight women. So far as this impairment is concerned, overweight is not a determining factor and warrants less prominence in our rating than heretofore. If anything, we should be cautious about applicants who are underweight at time of examination to determine if there has been an appreciable and significant loss of weight since the gall bladder attack, suggestive of the possibility of cancer. The relation of build to prognosis must, however, be left open for the present as one of the subjects for future investigation when a sufficiently large experience is available.

May I, in closing, urge upon you the desirability of pooling the experience of many companies on risks of this type. We shall, then, expedite the business of clearing up the several important elements in the selection of these risks about whose significance we are at present uncertain.

DR. FRASER—In considering the best way to place the discussion of Dr. Dublin's paper before you, we decided that it would be better to pick out a surgeon of ability and a medical man of ability and have the discussion taken from both points of view. We are particularly fortunate in having with us Dr. Russell S. Fowler, of Brooklyn, who will take the surgical end of the discussion. Dr. Fowler.

DR. FOWLER—Mr. President and Gentlemen: It is a pleasure and a profitable one to have listened to Dr. Dublin's paper. I appreciate the opportunity of discussing it.

The study of morbidity and mortality of gall bladder disease is a most fascinating one. It has engaged some of the most brilliant minds, both medical and surgical. From ancient times the disease has been baffling and attractive and controversial.

My own study of the disease dates from December, 1893, when while a third year student at P. & S. I experienced a most agonizing attack of gall bladder colic. Huntington's lectures on Anatomy and Prudden's Pathology and Curtis' Physiology were reattended and restudied with the interest which formerly was expended in the pursuit of good times which at that age and place was the habit of most of us. Even German was revived to a sufficient extent to read Hans Kehr of Halberstadt contributions and Ashoff's wonderful pathologic studies of the disease. This study has extended through my entire life. The disease with its ramifications has proved more interesting than golf or any other recreation. It provides problems in logic in history and in mathematics, and provides in the complexity which the complicated cases furnish, a most fascinating game requiring delicate skill and judgment unrivaled in operative surgery.

Material for Study—I have been particularly fortunate in that from the beginning of my surgical career I have had exceptional opportunities, 57 years of overlapping continuous hospital service, ten continuous years at the Brooklyn Hos-

pital, twelve complete years (over a period of thirty-six years) at the Methodist Episcopal Hospital and thirty-five continuous years at the German (Wyckoff Heights) Hospital of Brooklyn. These hospital years, together with cases furnished through the kindness of my medical friends who knew of my interest in the disease, have enabld me to study a comparatively large number of surgical cases of all kinds. I have been fortunate in being able to study gall bladder disease in its various aspects, both medical and surgical. This disease of all others must interest the medical clinician as well as the surgeon conjointly.

Definition of the Disease—Gall bladder disease is a chronic slowly progressive inflammation with a large incidence in relation to population, beginning in young adult life and gradually increasing in severity with markedly variable clinical manifestations, the latter so variable as to many times pass as negligible factors as regards health, the disease subject in a large proportion of cases to long periods of latency as regards clinical manifestations and in a small proportion of cases to sudden exaccerbations of acute inflammation. In the course of the disease the function of the liver is affected to a variable degree due to mechanical, nerve and inflammatory changes. Nearby organs are affected through lymphatic supply (pancreatitis, localized hepatitis) and through contiguity (adhesions, perforation) and through mechanical effects (pancreatic duct). Distant organs and mentality are affected through liver disturbance (jaundice, sluggish liver) and through the gall bladder acting as a focal infecting agent (kidney lesions, heart lesions, joint lesions). The disease is not only a wide spread disease but has limitless potentialities.

Incidence of Gall Bladder Disease—Our experience leads us to believe that gall bladder disease is even more wide-spread than Dr. Dublin's paper indicates for the following reasons: It is only in the last two decades that the knowledge of gall bladder pathology, before that time not widely recognized

and understood, has been better known so that we may consider autopsy findings as being minimum rather than maximum figures. Moreover, even now the gall bladder which I described in 1916 as Cholecystitis Catarrhalis Subacuta, the first stage in gall bladder disease, is perhaps not recognizable through its gross pathologic appearance by any except the most expert.

The Surgeon Looks at Gall Bladder Disease—Considering gall bladder disease as defined, only a very small percentage of cases require surgical intervention. It is estimated that of every 100 cases 80 either become quiescent or present such minor disturbances as not to interfere very much with the patient's well-being and therefore do not require surgical intervention. Of the remaining 20 there are perhaps 15 in whom operation is distinctly indicated and 5 in which it is a question of judgment as to whether or not operation should be performed.

If latency could be brought about by diet, medication or treatment other than operation it would of course be preferable.

In order to properly understand what I am trying to bring out in this discussion it is necessary to take up very briefly from the surgical standpoint what gall bladder cases may be treated without operation. Medical treatment is indicated in all acute inflammations of the gall bladder showing a tendency to subside with the exception of acute cholecystitis with gangrene, perforation, empyema and cholangitis. Medical treatment is also indicated in all chronic cholecystitis cases which tend toward latency and in hydrops of the gall bladder and in cholecystitis cicatrans not giving pain. Why is medical treatment indicated in these acute and chronic inflammations? In the acute because they are temporary and in both acute and chronic because they are not disabling, do not give the patient much discomfort and do not interfere to any great extent with his enjoyment of life nor his ability to earn his living.

Surgical treatment is indicated in acute cholecystitis which does not subside quickly, cholangitis both acute and chronic, gangrene of the gall bladder, perforation of the gall bladder, beginning carcinoma of the gall bladder, in all cases of chronic cholecystitis, of which there are many forms, in which health is affected over a period of time either through recurring attacks of pain or which cannot be brought to a degree of latency permitting of comfortable digestion and the pursuit of livelihood without too much interference.

There are two classes of surgical cases: those in which operation must be done at once in order to save life and those in which we have time to consider whether operation is advisable or not. In the first it is the physician and surgeon who must throw the weight of their experience in the balance to cause the patient to decide and must insist upon an immediate operation. In the second class there is time in which the case may be studied, discussed with the patient and a mutual decision arrived at in accordance with the degree of interference with the patient's comfort and livelihood.

In the first class are the cases of acute cholecystitis not rapidly subsiding, the cases of seropurolent chronic cholangitis, acute cholangitis, gangrene of the gall bladder. In the second class there are cases of beginning gall bladder carcinoma and the cases of chronic cholecystitis in which the patient's comfort and livelihood are being materially affected. I think that all physicians and surgeons can agree with this classification. Certainly in the first group we can all agree as these septic conditions are a direct and immediate menace to life. In the second group it is really up to the patient to give the final decision. He knows and he alone knows how much interference there is with his comfort and livelihood. We can, however, lay before such a patient his probable chances for cure or at least for the decided amelioration of his symptoms by operation and also the mortality of such an operation in skillful hands.

In many cases it will happen that in spite of recurring disabling symptoms the patient will so dread an operation as to refuse it when the attack has once subsided. Unfortunately many of these patients cannot understand that their chances of recovery and of cure are greatly enhanced if the operation is done between the attacks at a time when the minimum of inflammation is present, when they feel quite well and free from symptoms. If such patients are operated upon during an attack at a time when they will more readily submit to an operation on account of their desire for relief of pain we cannot, in many instances, do a radical operation which will almost certainly relieve them, but must content ourselves on account of technical difficulties and dangers in doing a drainage operation, thus leaving a gall bladder which, in at least 25 per cent. of the cases, gives subsequent trouble requiring operation with a higher mortality compared to operation in ectomy cases. Frequently if it is explained to the patient that an operation between the attacks minimizes the possibility of a second operation the patient will more readily submit. If, however, the patient will only submit during an acute attack then it is again up to the physician to decide whether such an operation should be performed. In this decision he will be aided by a study of the history as to the degree of discomfort and disability. We hesitate to make an emergency operation of such cases because we know how much better a result can be achieved by operation between attacks. Here let me interpolate a word of warning. The inflammation of the acute attack does not subside as quickly as the subsidence of the symptoms indicates. I have operated three or four weeks after the subsidence of all symptoms and have still found a very considerable amount of inflammation, so much so as to make ideal cholecystectomy of more technical difficulty than one would imagine. It is better to wait six weeks after a severe attack before operating if the condition of the patient will permit. It is certainly not waiting a sufficient time to

operate a few days following an acute attack as is the custom so often in our hospitals. Our objective should be to lower the mortality of gall bladder surgery to a point that will approximate the true mortality of the disease unoperated and by so doing convince our medical friends of the advisability of early operation.

In the cases in which we advise operation we base our advice on the degree of discomfort and interference with the patient's pleasures, comfort and livelihood. The symptoms need not necessarily be severe nor necessarily colics, they may be more in the nature of pressure sufficiently painful to aggravate the patient and to keep him from doing his work properly. Many such patients continue to work but their efficiency is impaired. Where patients present symptoms of nagging discomfort they should be advised that operation will cure or at least relieve them to a great extent. As a matter of fact patients presenting these symptoms between their acute attacks become worse and worse the longer they delay and finally must come to operation. The earlier the operation is done the better for them.

These remarks may not seem at first sight to be closely related to the discussion at hand but experience shows that if the above course is deviated from the final result as regards both mortality and morbidity will be increasedly worse. The history of what was done, when it was done and the pathology present, the degree to which the pathology could be overcome by operation, the potentiality for harm as to what was left are the most important factors in estimating the effect of the disease on the life span.

It is interesting for the surgeon to roughly work out the extent to which gall bladder disease requiring surgery impairs and shortens life. We start with the incidence of gall bladder disease to the whole population (life insurance figures may be entirely different as they involve but a small proportion of the population and as a rule do not have a sufficiently complete history of the disease but they could

be worked out accurately if a proper history of all cases could be obtained). The incidence to population we conservatively place at 60% (autopsy records). Of this basic 60% 80% either become quiescent or present such minor disturbances as not to interfere very much with the individuals well-being and therefore the question of surgical intervention does not arise. Of the remaining 20% of the basic 60% 75% present symptoms for which operation is indicated and the remaining 25% (5% of the basic 60%) are borderline cases in which it is a question of judgment as to whether operation should be performed or not. In this latter class also fall the cases refused operation not only because of insufficient indications but also those refused operation on account of having an operative risk out of proportion to the possible benefits and also those in which operation cannot be thought of on account of other disease conditions contraindicating operative interference of any kind. This class would in all probability present such a picture as not to be considered for insurance at all. We also rule out of consideration the 80% whose symptoms are negligible and for our present purpose of mortality study only the 15% who are properly surgical cases. We have seen by preceding paragraphs what constitutes a surgical case and what a medical case.

Immediate Mortality—What does a study of 1,206 of such operative cases show as regards immediate mortality? The immediate results are shown in Table I on the following page. All cases considered are personally operated cases.

There is a lot of food for thought in these figures. Note the tremendous difference in mortality in favor of operating in chronic inflammation rather than in acute. Note the better immediate results in inflammation limited to the gall bladder compared to the results when the disease has involved the ducts.

We can, I think, omit for the present purpose consideration of all cases except those allowing of cholecystectomy or

TABLE I

OPERATIONS: IMMEDIATE RESULTS

	Cases	Recovered	Died	Mortality (per cent.)
Cholecystostomy				
Acute	235	205	30	12.8
Chronic	17	17	0	0
	252	222	30	11.9
Cholecystostomy and Choledochostomy				
Acute	7	6	1	14.3
Chronic	5	5	0	0
	12	11	1	8.33
	264	233	31	11.7
Cholecystectomy				
Acute	278	260	18	6.5
Chronic	589	578	11	1.9
	867	838	29	3.33
Cholecystectomy and Choledochostomy				
Acute	19	14	5	26.3
Chronic	30	30	0	0
	49	44	5	10.2
	916	882	34	3.7
Choledochostomy Alone				
Acute	6	4	2	33.33
Chronic	11	9	2	18.2
	17	13	4	23.5
Cholecystogastrostomy				
Chronic	2	2	0	0
Choledochogastrostomy				
Chronic	1	1	0	0
Choledochoplasty				
Chronic	6	4	2	33.33
	1206	1135	71	5.9
Ops. in Acute Cases	545	489	56	10.3
Ops. in Chronic Cases	661	646	15	2.3
	1206	1135	71	5.9

cholecystostomy for the reason that cases which have advanced pathology outside the gall bladder should be considered as separate distinct risks of much greater morbidity and mortality than cases in which the gall bladder alone is involved, though the combined immediate mortality of all cases should be taken into consideration as regards the ultimate mortality of the disease.

Carcinoma cases can be put down as 2% of the surgical cases (the incidence of carcinoma to gall bladder disease considered surgically is, of course, higher than carcinoma to general population).

What then has been the end result of gall bladder disease limited to the gall bladder? This is shown in the following table which, leaving out the choledochostomies, cholecystogastrostomies, and choledochoplasties, shows the end-results in 979 cholecystectomies and cholecystostomies three to twenty-five years post-operative. Those patients relieved of all symptoms are called well; those who have an occasional mild indigestion or suffer mildly from over-eating and are immediately relieved by diet are classed as having mild symptoms; while those whose disabled liver, disabled from

TABLE II

	Cases	Per Cent.
Cholecystostomy	165	
Well	96	58.1
Showing mild symptoms	24	14.5
Showing severe symptoms	45	27.3
Total showing symptoms	69	41.8
Cholecystectomy	814	
Well	705	86.6
Showing mild symptoms	79	9.7
Showing severe symptoms	30	3.7
Total showing symptoms	109	13.4
Total Cases of Cholecystostomy and Cholecystectomy	979	
Well	801	81.8
Showing mild symptoms	103	10.5
Showing severe symptoms	75	7.7
Showing symptoms	178	18.2

the long continuance of the infection before submitting to operation, require considerable care in diet, are classed as having severe symptoms.

Here again is plenty of food for thought. The results are fair but how much better they would have been had the patients been operated on before the liver was damaged so extensively as to require treatment even after the infecting focus was removed, and before the inflammation had resulted in adhesions, stricture of ducts, perforation, gangrene, peritonitis, cholangitis, liver insufficiency, hepatitis, liver abscess; distant infections from the local focus such as myocarditis, renal insufficiency, pulmonary infections, joint complications; involvement of neighboring organs through extension of the inflammation such as pancreatitis, perforation into the stomach, duodenum or colon, adenitis causing pressure on the common duct either beneath the liver or at or near the ampulla of Vater; carcinoma of the gall bladder following long-continued chronic irritation to say nothing of the discomfort and disability from the at first localized disease.

We are considering the 15% representing the cases in which surgical treatment is indicated. We have an immediate average gross mortality of 5.9% (ranging from 0 to 33⅓% according to pathology found at operation) including the desperate cases. We have an ultimate average severe morbidity percentage of 7.7% (ranging from 3.7% in ectomy cases to 27.3% in ostomy cases) which percentage must be considered as a minimum.

From a study of secondary operations made after cholecystostomy we find we do not have to add a further mortality table. A study made years ago showed this mortality and morbidity to be larger than that of primary ectomies.

Variations in technique make a difference in mortality and morbidity as shown in a study of ectomies with and without drainage (Table III).

A comparison of these statistics shows that the mortality percentage is in favor of cholecystectomy with drainage.

This is all the more remarkable when we consider that cholecystectomy with drainage was done in the more advanced cases while cholecystectomy without drainage was done in the simpler cases.

As to morbidity there is a marked difference in each class in favor of the cases which were drained and this is again

TABLE III

MORTALITY AND MORBIDITY COMPARISON OF ECTOMY WITH AND WITHOUT DRAINAGE

MORTALITY			WITH DRAINAGE MORBIDITY								
								Symptoms			
Number	Died	Per Cent.	Examined	Well	%	Mild	%	Severe	%	Total	%
543	15	2.7	406	365	94.8	10	2½	11	2.7	21	5.2
			WITHOUT DRAINAGE								
240	8	3.33	134	120	89.6	8	5.9	6	4.5	14	10.4
783	23	2.93									

TABLE V

Died 5 Years Post-Op.	Cancer of breast	1
	Erysipelas	1
	Insanity	1
Died 6 Years Post-Op.	Myocarditis	2
	Cancer of pancreas	1
	Cause unknown	1
Died 7 Years Post-Op.	Pneumonia	1
	Cause unknown	3
	Cancer of stomach	1
	Cancer of liver	1
	Myocarditis	1
	Cancer	1
Died 8 Years Post-Op.	Cholangitis	1
Died 9 Years Post-Op.	Nephritis	1
	Pneumonia	1
Died 10 Years Post-Op.	Cancer of liver	1
	Stricture of common duct	1
Died 13 Years Post-Op.	Duodenal ulcer	1
Died 15 Years Post-Op.	Cancer of pancreas	2
Died 16 Years Post-Op.	Cancer of breast	1
Died 17 Years Post-Op.	Apoplexy	1
		25

the more remarkable as the drained cases undoubtedly showed more advanced pathology. When we find, as we do in this analysis, that in a series of 240 cases the percentage of deaths is greater than in a very much larger series of drained cases more severe in character, and when we find that in the analysis of the final results the drained cases show a considerable superiority in percentage of cures over the undrained, we must conclude that in spite of the temptation not to drain, it is wiser to drain.

TABLE VI

There are 9 cases which are known to have died less than 5 years post-op., as follows:

2 Months	Myocarditis	1
	Cardiac dilatation	1
4 Months	Pneumonia	1
9 Months	Phlebitis and Pyemia	1
1 Year	Aneurysm of hepatic artery	1
	Cirrhosis of liver	1
	Cause unknown	1
	Accidental gas poisoning	1
2 Years	Cause unknown	1
		9

TABLE VII

Number of Cases Considered....34—		**Connection With Gall Bladder Disease**
Cause unknown	6—	6 unknown
Accident	1	21 not connected
Insanity	1	
Cardia, circulatory	8	
Erysipelas	1	
Nephritis	1	
Pneumonia	3	
Cirrhosis of liver	1	
Cancer	4	
Duodenal ulcer	1	
Cancer of pancreas	3	7 direct connection
Stricture of common duct	1	
Cancer of liver	2	
Cholangitis	1	
	34	

MORTALITY AND MORBIDITY COMPARISON OF OSTOMY AND ECTOMY SEPARATELY AS WELL AS COLLECTIVELY IN RELATION TO PERCENTAGES OF POPULATION

It will be illuminating to consider the ostomy and ectomy cases separately as well as collectively.

Ostomy acute and chronic mortality	11.9%
Ostomy acute and chronic morbidity	27.3%
Ectomy acute and chronic mortality	3.33%
Ectomy acute and chronic morbidity	3.7%
All cases ostomy and ectomy acute and chronic mortality	5.27%
All cases ostomy and ectomy acute and chronic morbidity	7.7%

Using these figures which are average figures for the skilled operator and therefore minimum percentages one can figure out the expected mortality and morbidity percentages for both the entire population or the proportion of the population having gall bladder disease as regards surgical treatment. The percentage of both mortality and morbidity would be small in both instances as only 15% of all gall bladder disease as we have seen fall in the surgical category. It would still be small even though borderline cases of 5% be included; or whatever classification we make as regards cases requiring medical or surgical treatment this would not affect the gross percentage 20% requiring some kind of treatment. At most in a properly diagnosed case the final mortality of the disease should not exceed 1.054% and the morbidity should not exceed 1.54%.

It will be interesting to note how these roughly arrived at figures compare with the better detailed study made by the authors of the paper. As I am not familiar with the Insurance methods of arriving at mortality and morbidity statistics I cannot make the comparison myself.

What is of equal importance is the length of life after operation for gall bladder disease.

I have studied 1,489 histories of patients in which I personally operated on who were still alive at least five years following operation (Table IV). This list is incomplete as hundreds of cases have not been traced.

In addition to these cases 25 are known to have died (Table V) at periods ranging from five to seventeen years post-op. Nine additional cases died two months to two years post-op. (Table VI). The cause of death in relation to gall bladder disease is shown in Table VII. I do not think these tables are very valuable to you but they are presented for what they may be worth.

TABLE IV

ECTOMY AND OSTOMY CASES
ALIVE 5 TO 25 YEARS FOLLOWING OPERATION

Ectomy,	Acute,	last	reported	alive	5	years	post-op.	.....	259	cases
"	"	"	"	"	10	"	"	.....	37	"
"	"	"	"	"	15	"	"	.....	15	"
"	"	"	"	"	20	"	"	.....	2	"
"	"	"	"	"	25	"	"	.....	2	"
Ectomy,	Chronic,	last	reported	alive	5	years	post-op.	.....	641	cases
"	"	"	"	"	10	"	"	.....	180	"
"	"	"	"	"	15	"	"	.....	72	"
"	"	"	"	"	20	"	"	.....	16	"
"	"	"	"	"	25	"	"	.....	0	"
Ostomy,	Acute,	last	reported	alive	5	years	post-op.	.....	186	cases
"	"	"	"	"	10	"	"	.....	36	"
"	"	"	"	"	15	"	"	.....	23	"
"	"	"	"	"	20	"	"	.....	5	"
"	"	"	"	"	25	"	"	.....	1	"
Ostomy,	Chronic,	last	reported	alive	5	years	post-op.	.....	9	cases
"	"	"	"	"	10	"	"	.....	0	"
"	"	"	"	"	15	"	"	.....	2	"
"	"	"	"	"	20	"	"	.....	3	"
"	"	"	"	"	25	"	"	.....	0	"

Making a total number of operative cases alive at least 5 years post-op. of 1,489

I would judge that the cases considered in Dr. Dublin's paper fall in the 20% class who present sufficiently severe symptoms to require treatment of some kind. What a surprise to find that the total number of deaths (159), not necessarily from the disease, is so large and that the num-

ber of deaths among men are two times the number among women. The number of deaths attributed to the disease are 11, practically equally divided between men (5) and women (6) of which ectomy was 2, ostomy 2, operation not specified 2 and medical 5, practically equal for medical or surgical treatment so far as *direct* deaths from gall bladder disease are concerned. From *indirect* causes probably but not necessarily certainly connected with gall bladder disease and excluding cancer we find 28 deaths about equally divided, men (16) and women (12) and more (17) occurring after surgical treatment than after medical treatment (11). Personally I object to classifying the disease into inflammation with stones and inflammation without stones as I believe the stone to be a by-product of the inflammation. The reason that more deaths occur in cases showing stones is because the presence of stone shows the case as having progressed further in its progressive chronic inflammation.

Combining the non-cancer direct and indirect deaths we find 39 about equally divided between men (21) and women (18) of which 23 were surgical treatment cases and 16 medical treatment cases.

I have excluded cancer. Why? Cancer should be considered in my judgment as a disease by itself and not together with inflammation of any type, for reasons which are not germane for the subject under discussion this afternoon.

Can we compare these figures with the clinical figures which I have presented? No. Why? Because the insurance cases are completed cases all of which are definitely under control while the clinical cases are so often lost track of. The clinical cases, however, have the advantage of a more definite and complete history, particularly as to the pathologic conditions present at the operation, so that a more definite connection can be established between morbidity and final mortality and a more definite prognosis be made in a given case as to the course which that case will pursue. This is a decided advantage and almost a sine qua non in

insurance cases and should be made a matter of very definite record in accepting gall bladder risks. Therefore it is necessary that all records be consulted before such a risk is accepted. This of course is true in all diseases to a certain extent but more decidedly true in gall bladder disease, as gall bladder disease is such a common wide-spread disease capable of causing so much morbidity.

It is certainly a matter worthy of further study to determine the added percentage of risk, if any, caused by gall bladder disease. I am sure that many physicians, surgeons, hospitals and patients would be glad to co-operate if the matter were properly presented so that it should not be too difficult to collect statistics in 100,000 cases which had either died or completed a twenty year period.

Such a study of the life history of a disease as wide spread as gall bladder disease could be made of much value to the public.

In closing, let me again thank you for the privilege of discussing before you the interesting subject so ably presented by Dr. Dublin.

DR. FRASER—The medical end of this discussion of Dr. Dublin's paper will now be taken up by Dr. Burrill B. Crohn of the Mt. Sinai Hospital, New York City. Dr. Crohn.

DR. CROHN—Mr. Chairman and Gentlemen: Reading and studying Dr. Dublin's paper has been a pleasure and an education, an education to see the very clear, scientific, impartial way in which such statistics have been collected. It is rather unusual to have such a follow-up in medical practice and have such an impartial group of figures presented. Medical follow-ups in general are rather carelessly gotten together and their value depends more or less on the personality of the individual who presents them, whether medical or surgical.

I am envious of Dr. Fowler's mass of statistics because I present no statistics but prefer to discuss the subject from the purely clinical or medical angle. One of the difficulties en-

countered in a study of gall bladder disease is the almost complete absence of any mention in the literature of a medical follow up of gall bladder disease. In all the literature, only one paper has been written on the medical follow up of gall bladder disease, and that is the recent work of Blackford on the Pacific Coast, in which he followed up 200 cases over a period of years. You can con medical literature over and over and you will not find another follow up of the medical history of gall bladder disease, showing the tremendous field which is still open.

Now, discussing more intimately Dr. Dublin's paper, I think one must put on an achromatic lens in looking at such figures, because there are two or three facts that I would like to point out which, I think, alter to a certain extent the clinical acceptance of his figures.

First of all, Dr. Dublin's figures are based upon past gall bladder disease; 80% were operated. If one attempted to compare the actuarial figures with clinical figures, Dr. Dublin's would seem like a follow up of operated cases. A comparison of the medical follow up with the clinical follow up of the physician should take into consideration the fact than the disease is actually present. Of course, you who are interested in life insurance statistics must know that you have carried at all times medical gall bladder cases which you are not recognizing if the incidence of gall bladder disease is as high as is claimed in medical statistics. If one accepts Mentzer's autopsy figure of 66%, then you must assume that 66% of all your risks have gall bladder disease, which is far above anything that appears in the questionnaire which a man signs when taking life insurance, and you must at all times be carrying huge numbers of gall bladders of which you have no knowledge and which you have no means of being apprised of. However, I don't know that Mentzer's figure of 66% is accurate.

I wish to undermine a certain amount of confidence in the whole statistics of gall bladder disease, which in a way I will explain in a moment. Mentzer says 66% at autopsy had cholecystitis. The trouble was started with strawberry gall blad-

der, which came out of the famous Mayo Clinic, and included in such categories, pericholecystitis, deformities of the gall bladder, chronic atrophic cholecystitis, and you get an entirely wrong conception of what constitutes clinical gall bladder disease. Those are not diseases. I can not conceive of strawberry gall bladder as clinical gall bladder disease. I am sure that in statistics of operated gall bladder cases you are including a very high percentage of cases which were operated under a misconception, or what to me is a misconception of what constitutes gall bladder disease.

For instance, 25% of the population have gall stones. Yet, according to statistics, only 6% of these cases have gall bladder inflamation of clinical manifestation. It is quite obvious that if only 6%, or 1 out of 10 cases of gall stones complain, the other 9 are silent. The fact of the matter is that I know you are carrying a large percentage of stones and a large percentage of gall bladder disease in the population who are not complaining of any symptoms at all. The question is, what happens to such cases? You come back again to the necessity for a follow up of medical cases. In other words, my conclusion in that respect is that there is a high incidence of silent gall stones or innocent gall stones. You may recall the famous statement that it was not the gall stones that were innocent; it was the doctor who was innocent—and with that statement gall bladder surgery was let loose on any thing that looked like it.

The fact of the matter is that there is such a thing as silent gall stones, and I learned that recently as I will tell you in a moment, because if you choose for operation only such cases of well-defined gall bladder disease with stones, then the figure quoted of cure after operation of 83% to 100% is probably accurate. If the case has stones and cholecystitis, there is every chance in the world that you will have a perfect result. Now as soon as you come to the acalculous cholecystitis, you have a different field entirely, because the acalculous cholecystitis includes not only true chronic or acute cholecystitis, but also includes a huge percentage or proportion of neurotic women with vague abdominal symptoms and vague pains in the gall bladder region

and the indications are awfully mild and awfully faint. Many times I have seen people operated on for mild complaints, the physical examination disclosing only a mild point of tenderness over the Murphy point.

Now, your acalculous cholecystitis figures show immediately that the percentage of cures has dropped to 70%, and the only way to understand the difference between 100% of cures in calculous cholecystitis and the 70% in acalculous cholecystitis is to realize that among the acalculous cholecystitis you have included possibly 30% of unnecessary indications for operation. And my hands are not clean. I have done it myself. I have done it with my eyes open and I will do it tomorrow knowing and feeling the way I do. I am bound to mistake the diagnosis.

Can stones ever be silent? I learned that about two years ago from a patient, a woman of the type that come in the office with vague abdominal symptoms. A routine X-ray showed gall stones. She had vague abdominal pains, discomfort, dyspepsia and constipation. I did not have much hesitation in suggesting that the patient be operated upon and she was immediately operated upon and her gall bladder removed. So far as the surgeon and I were concerned, everything was perfectly satisfactory. However, within two or three weeks after the operation she went back to her old complaints again, vague abdominal pains, constipation, etc., and then began vomiting, typical vomiting on an empty stomach, which I knew to be functional and neurotic. I became interested in the psychic side of the patient, something I should have done before, and on making my own psychoanalysis, discovered a topsy-turvey complex which made me realize that I had removed the gall stones of that woman because the X-ray showed gall stones and caused me to jump to the conclusion that because she had gall stones, the abnormal symptoms were due to the gall stones. I then realized that in a large percentage of cases the gall bladder is removed because the X-ray shows gall stones when the actual cause of the complaint is fundamentally an abdominal neurosis, and that is a very unsatisfactory type of cases to handle in practice. That

type of cases gives you your 30% of failures in acalculous cholecystitis due to the unnecessary removal of the gall bladder because of a deformity in the outline of the gall bladder or because of stones.

The same point holds for the causes of death. Dr. Dublin's follow up of gall bladder cases indicates a fairly high percentage of deaths not attributable to gall bladder pathology in itself. According to Dr. Dublin's paper, a large percentage of deaths were due to a non-malignant or malignant condition of the alimentary tract. There again it is quite obvious that the gall bladder was removed as a result of a mistaken diagnosis and the patient had latent carcinoma or a carcinoma so small that it was overlooked at exploratory, and I am sure that you will find that most of the malignancy deaths occurred within two years of the gall bladder operation.

I recall an analysis of figures of gastroenterostomies following gastric ulcer, the largest percentage of deaths being due to malignancies. Most of them were discovered after two years, which, of course, convinced me that at the time of the gastroenterostomy the lesion was considered innocent, but actually the lesion was malignant. As a result, these cases may alter the figures and one must read statistics with always that in mind.

For instance, Graham notes the fact that 14% of the cases coming in for gall bladder operation gave previous histories of appendectomies, probably for chronic appendicitis.

The higher mortality in men calls for interpretation. Most of the men operated are really sick, while a large percentage of neurotic women are included in the category of chronic cholecystitis. Functional cases give a low mortality. They are easy cases to operate, no complications, no infection, have a low mortality. A man has got to be sick before he submits to an operation. In a man it is more serious and you can practically rule out the neurotic individuals.

What are the dangers and possibility of avoiding operation? Well, it is almost impossible to say, because the only figures that are practical ones are Blackford's, and it is remarkable that in

his small follow up the mortality due to gall bladder disease is only 1%. Your surgical mortality in the hands of experienced surgeons, like Dr. Fowler, is 1.9%. As a general rule the mortality for gall bladder operation is between 2% and 5%, probably near 5%. We know from the statistics gathered from large groups of surgeons all over the country that it approaches near to 5%. Dr. Fowler rightly points out the fact that the statistics are based on gall bladder statistics gathered from all over the country and therefore the surgical procedure is performed in every hamlet, village and city and there must necessarily be a huge difference in the ability and efficacy of the surgeons.

Of the 90% of silent cases of gall bladder disease and gall stones, which we are carrying, very few run into surgical complications. I just want to point out to you that there is no great urgency about rushing a gall bladder case to the operating table even after the indications are present. Dr. Dublin shows very rightly that it is not the stones but the cholecystitis which is the deciding point in operation.

I must compliment the Association and Dr. Dublin for the type of figures shown and for the remarkable way in which he has placed the risks in the different classes. The follow up shows that the mortality for standard risks is 96% and that in the Intermediate class, which, I understand, has a higher rating, it is 121% and in the Special class the mortality is 143%. It is quite obvious that you life insurance men are excellent clinicians as well as statisticians.

I must say again that it has been a privilege to discuss the paper and hear the papers read, and I appreciate the clear, concise, impartial way in which these figures have been gathered and have been shown. I thank you.

Dr. Fraser—We are peculiarly indebted to Dr. Crohn in that he is appearing before us at the time of bereavement in his family. His sense of duty, however, was so high that he sunk his own personal feelings to appear before us on the day of a funeral in his family. We thank him.

The discussion on this paper will now be closed by Dr. Jimenis of the Metropolitan Life Insurance Company.

Dr. Jimenis—Mr. President and Gentlemen: I haven't very much more to add. We are extremely indebted to Drs. Fowler and Crohn for a very helpful discussion. I took a look at this study from a different angle, looked at it backwards and got up some death claims of people who had died of gall bladder disease, including carcinoma of the gall bladder or liver, and I found that in such a group there was a history of gall bladder disease in one third of them. I must amend that. It was not only gall bladder disease but also disease of the digestive tract as a whole. That is roughly what we have to work with.

Of course, we are limited in our selection. We can not go to the length that the clinician can, but even working as we do, one third of these deaths (they are the people who died from gall bladder disease) had previous histories of digestive tract disorder.

The inconsistencies in the report, as Dr. Dublin pointed out, can only be cleared up by studying a larger group. Dr. Dublin stands ready to cooperate with any group for this purpose.

We have now accumulated quite a bit of information respecting the interval that should elapse before we can issue standard insurance on cases of unoperated gall bladder disease. Most companies, I think, withhold standard insurance in non-operative cases for at least four years. I think we are about as liberal as any in this regard.

The Medico-Actuarial Mortality Investigation in 1914 found no difference in the unoperated cases between those in whom the illness had occurred within five years and five to ten years previous. A similar group in the Medical Impairment Study shows only a slight improvement at five years. In our own unoperated group there was no improvement after five years; in fact, it was worse.

The operated group, however, does show an improvement in mortality with advancing years.

Of course, we have all realized for some time that the drainage cases were the worst of the lot. I am not sure that we have realized just how bad they are, but to my knowledge, none of us has ever taken into consideration the sex of the applicant, and this, I believe, we should do.

DR. FRASER—The next paper on the program will be that of Dr. Edward S. Dillon, of the Penn Mutual Life Insurance Company, on the "Clinical Experience with Diabetes Mellitus Applied to Life Insurance." The discussers will be Dr. Elliott P. Joslin and Dr. Donald B. Cragin, of the Aetna. We are about to reverse the usual procedure, and with the permission of Dr. Dillon, we are going to call upon Dr. Joslin to start the discussion and then Dr. Cragin and then Dr. Dillon to sum up with remarks regarding his paper. I will now call on Dr. Joslin.

DR. JOSLIN—Dr. Fraser and Gentlemen of the Life Insurance Medical Directors Association: Dr. Dillon's common sense paper upon his clinical experience with diabetes applied to life insurance cannot help being important to the Association of Life Insurance Medical Directors whose companies spend $18,500,000 annually in death claims for diabetics and attribute to diabetes the cause of 9.3 per cent. of all rejections on medical grounds. The mere fact that diabetes appears so frequently upon the programs of this medical body is further proof, if any were needed, of its importance as an insurance problem.

Dr. Dillon's opening paragraph like the concluding paragraph of Dr. Rabinowitch's paper last year emphasizes fundamentals in the detection and diagnosis of diabetes mellitus and of allied suspicious glycosurias. Diagnosis should be based on a comprehensive basis—(1) the reports of the laboratory, (2) the family history, and (3) the symptoms and signs as disclosed by examination of the patient. One should utilize all these sources of information. (1) The demonstration of sugar in the urine—considering the patient to be diabetic until proven otherwise; (2) a study of the heredity;

(3) loss of weight and other symptoms characteristic of the disease.

These tests alone may exclude too many future, good policy holders and it is because of this fact that various laboratory frills—procedures—are adopted to salvage for insurance a goodly percentage of those discarded, but none the less never should one disregard glycosuria, heredity and symptoms in the selection of a case.

Perhaps diabetics are still uninsurable, as Dr. Dillon says, but I have no hesitation whatsoever in saying (a) that they are four times less uninsurable than they were thought to be in the Naunyn Era, 1898-1914. Then my cases died with a duration of 4.8 years, today I am convinced the average individual developing diabetes this year will live 20 years. People generally are thinking and acting altogether too much on the old duration. (b) The average diabetic develops his disease at about 50 years and if he is destined to live 20 years, you will soon need to think twice before rejecting him *carte blanche,* because about how long will the average man at 50 live anyway? (c) I have more than 170 living children, i.e., they developed diabetes under the age of 15 years, who have had diabetes 10 or more years. And the new diabetic child, and of course adult, is receiving far superior treatment to that in 1922 when insulin was first used. (d) Two other factors are of real importance, namely, diabetes is recognized earlier today than formerly, and treatment is therefore far more promptly instituted. Insurance and health examinations are responsible for that. And finally, milder cases are detected and indeed so mild that real difficulties of diagnosis arise. Therefore, think twice before rejecting each one of the 400,000 living diabetics in the United States, because you may do better caring for those you know about than to take a chance with the 2,000,000 now living as yet unrecognized who are to die of diabetes. Don't neglect to reap a harvest a few years hence of all the insurance propaganda you have planted in the lay mind about diabetes and its treatment.

An example of such propaganda is the diabetic number of the Commonhealth, published by the State of Massachusetts. I have provided a copy for each of you, chiefly with the idea that it may stimulate others, likewise interested in diabetes, to induce their State Governments to follow the example of Massachusetts. I took especial pains in arranging that our group contribute to this State publication, because I knew it represented the idea expressed to me years ago by Dr. Augustus I. Knight for a popular text on diabetes at little cost. The series of articles by Dr. Dublin and Mr. Marks in which I am cooperating is not of a popular character but I hope they will be found of real help to this group.

Above I referred to the duration of the diabetic's life in the Naunyn Era as 4.8 years. That represented the duration of life of those seen by me who died in that period 1898-1914. The true duration of the entire group was much longer and approaches 10 years. Indeed about 5 per cent. of the total number are still living. So too, in the Allen Era the true duration for the entire group will probably be about 15 years. The Era closed in 1922, but over 20 per cent. of the cases are now alive.

Insurance companies pride themselves on their follow-ups, their end-results and so do I. But you follow up your accepted cases whereas I follow up true diabetics and the glycosurics. Today I can report definitely on a few groups and partially upon others, but eventually Dr. Dublin, Mr. Marks and I will report upon all, and the follow-up will surely include 97 to 98 per cent. of all groups.

Pentosurics. Four cases have been recognized among the 13,000 coming for glycosuria. All are alive. Undoubtedly we have missed a few more.

Renal Glycosurics. My cases will answer Dr. Cragin's question of last year, although perhaps my definition of the group is sharper and narrower. A renal glycosuric to me is a symptomless glycosuric, whose urine always shows sugar,

whose blood sugar is always normal, who has been observed for a considerable period. The percentage of sugar may be 5 per cent. or more, but is generally less, yet even fasting specimens contain sugar. There are 29 of these. All have been traced and all are alive save one, a little boy, who was killed in a coasting accident. The longest duration was 40 years and the average of the first 21 is 13 years. Not one has changed to a diabetic.

Now for the follow-up of the total unclassified glycosurias in the Naunyn Era, 1898-1914. The data are not complete, but thus far 5 per cent. of those I definitely thought were non-diabetics and 25 per cent. of those I believed to be potential diabetics, suspicious cases, have developed diabetes. For the Allen Era, 1914-1922, the percentages for the two groups are 13 per cent. and 10 per cent., respectively. I am at a loss to explain why twice as many of these I thought to be non-diabetics have changed to diabetics and less than half of those called suspicious have changed as compared with the Naunyn Era. Sugar tolerance tests were not common prior to 1922 and so cannot be blamed for the confusion. This naturally leads me to the discussion of the diagnosis of diabetes with the help of blood sugars and the sugar tolerance tests.

Capillary blood sugars I like in the treatment of diabetes in children and during the treatment of diabetic coma and following surgical operations, because capillary blood sugars save the veins for therapeutic procedures, but I do not like capillary blood sugars for diagnosis. They are more susceptible to error in the hands of the average technician. Whereas capillary and venous blood sugars are identical fasting, they differ widely from one another after food. This holds especially in non-diabetics in contrast to their behavior in severe diabetes where they closely approximate one another. I do not know the difference in the peak readings after food—perhaps one should add 30 milligrams, i.e., instead of taking 170 (0.17 per cent.) as a boundary line, take 200 (0.20 per

cent.). Personally I do not trust capillary blood sugars, but the error is all on the safe side for the insurance companies. You have asked me for my opinion and therefore I state it. And now follow other opinions which I am asked to give in answer to questions submitted.

A sugar tolerance test should be viewed as a whole just as the diagnosis or rather acceptance of a risk should be viewed as a whole. I am requested to state, if I had only one blood sugar test, which I should prefer and unhesitatingly I reply the peak at one-half or one hour intervals, next the fasting blood sugar, which of course is not at all conclusive because children and mild diabetics often show normal fasting values, and last of all the 2 and 3 hour values. Experience with surgical patients, who will vomit orange juice and ginger ale when they reach the operating table at 3 and even 4 hours after taking such soluble carbohydrates, convinces me that you can't tell when the food is being absorbed.

Tolerance tests anyway are deceptive. In a doubtful case I always try first the effect of a good square meal, such as so many of you ate today and should the patient show sugar in the urine and a blood sugar of 0.17 per cent. I would put him down as a diabetic. If less than 0.17 per cent. I would give a fasting sugar tolerance test with all the precautions so admirably described by Dr. Rabinowitch last year.

DIAGNOSTIC SUGAR TOLERANCE TESTS (Venous Blood)

Classification	Fasting Per Cent.	½ Hour Per Cent.	1 Hour Per Cent.	2 Hours Per Cent.
Safe	0.12	0.16	0.16	0.12
Suspicious	0.13	0.16	0.16	0.13
Diabetic	0.14	0.17	0.17	0.14

For so many years my cases have been classified on the basis described in the above table that I shall hesitate to change it. I prefer to learn the outcome of the group and think that will be more useful than to alter the basis. Others can report results with their methods and in that way the

preferable classification can be achieved. The one figure which I am most inclined to change at the present time would be the peak value, but I have no great inclination to alter it to 0.18 per cent. With such a figure more diabetics would be missed in contrast to my present plan with 0.17 per cent. which may wrongfully include too many.

The financial aspect of the diabetic problem is serious. The insurance companies do well to strive for the prevention of the disease by combatting obesity. That is always safe and a forthcoming article in the series by Dr. Dublin, Mr. Marks and me will show upon what a solid basis of fact such efforts can be based. Heredity is also an important consideration; indeed, it is the most important factor in the etiology of the disease, but the patients will take care of that to a large extent. It is proper to urge all diabetics to search for new cases among their relatives. Already they are beginning to do so. Don't say much about sterilization, because the patients will look out for that too. Remember that about one in four of the inhabitants of the United States have a diabetic element in their family. Although 100 per cent. of the children of two diabetics should develop the disease, in reality only 44 per cent. will do so, because the others will have died from other causes before the year at which they were supposed to develop the diseases.

The complications of diabetes cost. An attack of coma represents an expenditure of $100, a sore toe on the average costs fully $300, and tuberculosis, which is two to three times as frequent in diabetic adults as in non-diabetic adults and 13 times as common in diabetic children, is equivalent to $1,500 a year. Obviously we must arrange to treat our diabetics in a less expensive manner.

Summer camps meet the needs for diabetic children. At less than half the expense of hospitalization we cared for 115 children at four camps last summer and this year we plan to attempt extending camp privileges to a small group between 15 and 30 years of age. When a diabetic child goes

to a camp he receives good diabetic care, a change and a good time, and his mother has a vacation.

DR. FRASER—Because of the interest insurance companies have shown in regard to glycosuria and the value of the glucose test, we are certainly very much indebted to Dr. Joslin for his masterly speech. The second discusser will be Dr. Donald B. Cragin, of the Aetna Life Insurance Company. Dr. Cragin.

DR. CRAGIN—Dr. Fraser and members of the Medical Directors Association: My congratulations to the Penn Mutual and Dr. Dillon in getting down to clinical investigation of end results in relation to the insurability of risks. I have felt for some time that there is a possibility of a medico-actuarial investigation getting out of hand unless based on a solid clinical foundation. After all, more solid fact is to be learned at the autopsy than in the diagnostic clinic and we could well make Dr. Dillon's paper serve as a model for the future. It is important to know what to expect for the future of any substandard case or in cases with past impairments. Candor compels me to state that in the wild early days of the disability income clause our company was earnestly engaged in giving this benefit to cases with a past sugar record, not cleared by tolerance tests, and even in cases which were rated as at least mild diabetics or "suspicious". Fortunately, our sanity returned before much damage was done but we have at present a few diabetics living in luxurious ease on monthly checks to remind us of our folly.

Studies like the one under discussion would have been a red flag.

As most of you know, our company is using the tolerance test rather freely. We have watched our results carefully and are still convinced that our method is safe; but we are constantly marveling at what we find. Take, for example, the question of diabetic family histories. I tentatively suggested in 1931 that the figures of some clinics in regard to familial diabetes were too low. A brief analysis of our own records showed about 24% of true diabetics having had a member of the family similarly ill. Most

clinical figures were much lower. Those of you who have followed the recent papers on this topic will have noted the rapid jump in the percentages. Dr. Dillon has touched on the hereditary traits.

We are suspicious of overweight combined with certain racial connections and frequently pick a diabetic on our tests in this group.

It is naturally impossible for me to discuss this paper from the clinical standpoint as I have no facilities for the same and particularly so in view of the clinical eminence of the preceding discussor.

A brief summary of our experience with the tolerance test in regard to its workableness is here given:

From June 1, 1927, to June 1, 1934, we called for 4,959 sugar tolerance tests. 3,280 were completed. The not-completed were 1,679. Of the completed tests 641 were rejected; 213 were given Class A; 226 Class B; 34 Class C; and 2 Class D.

In what we call our sugar tolerance series—that is, those cases in which a sugar tolerance test has been made—we have had two deaths from diabetes. One of these was examined in the early days of the test (1927) when we allowed the applicant to feed himself a test meal. The fasting blood was 140. At the end of 1½ hours the blood sugar was 140. Previous record of sugar findings in 1926. We had our warning in the fasting blood. Two years later pronounced diabetic.

The other was taken in 1928 (100 grams glucose) and at the end of 1 hour and 35 minutes the blood sugar was 130. Family history negative but sugar had been found 3 months preceding the examination. The fasting blood was 80 mgs. Diabetes was diagnosed in 1932. Acute onset.

As a matter of fact, most of our diabetic deaths occur in Group Conversions in which we have no privilege of examination. (Over 60%.)

A complete study of our substandard diabetic mortality (620-627) is appended.

SUBSTANDARD EXPERIENCE ON SUGAR CASES

(620–627)

Years of Issue 1924-1932 (traced to anniversaries in 1933)

BY NUMBERS

Class	Years Exposed	Actual Deaths	Expected Deaths A. M. Select Table	Ratio	Expected Deaths S. S. Table	Ratio
A	648	12	6.2	193.5	7.3	164.4
B	3,479	36	38.6	93.3	54.1	66.5
C	225	7	3.4	205.9	5.9	118.6

BY AMOUNTS

Class						
A	6,258,000	130,000	70,878	183.4	82,856	156.9
B	32,468,000	388,000	355,014	109.3	498,087	77.9
C	2,580,000	80,000	38,658	206.9	65,719	121.7

Again, I want to express my appreciation of this excellent contribution and trust it is the forerunner of many more end-result investigations. Perhaps we can venture to suggest a follow-up on rejected cases. We have tried this in a few instances and the results were gratifying and illuminating. I trust this will be given consideration.

DR. FRASER—I will now call upon the author of this paper, "The Clinical Experience with Diabetes Mellitus Applied to Life Insurance", Dr. Edward S. Dillon of the Penn Mutual. Dr. Dillon.

CLINICAL EXPERIENCE WITH DIABETES MELLITUS APPLIED TO LIFE INSURANCE

EDWARD S. DILLON, M. D.

The mortality rate of diabetics, in the experience of life insurance companies, is so far above the rate of normal individuals that no provisions for the extra mortality can be made on any plan, and diabetics are, therefore, uninsurable. A history of glycosuria or the finding of sugar in the urine at the examination is nearly always the source of the suspicion that a given applicant may be diabetic; but a family history of diabetes, overweight or recent loss of weight may serve to strengthen this suspicion.

In the experience of life insurance companies with cases showing glycosuria, the ratio of the so-called benign cases to those in which the ability to utilize sugar is definitely diminished is much higher than in the experience of most practicing physicians. The matter of selection is, therefore, of prime importance; and for this purpose the blood sugar tolerance test has been used extensively. Many excellent papers on this subject have been presented before this Association in previous years; and, therefore, no further reference to it will be made. The problem of diabetes, however, is frequently presented in connection with disability claims and in connection with lapsed policies where the insured have developed diabetes after the policies were issued.

In recent decades the incidence of diabetes has been steadily on the increase. Part of this increase is only apparent. Many cases are now being detected which formerly would have been overlooked. Routine urinalyses are more frequently made by practicing physicians. Many cases are being discovered by life insurance examination and routine health surveys. The larger part of the increase, however, is real, and the causes will be briefly mentioned.

While diabetes may begin at any age, more than two-thirds of the cases begin at ages over forty. In the two decades from 1910 to 1930, the portion of our population forty years of age and over increased from twenty-two millions to thirty-six millions, an increase of 63.7 per cent.; while the portion under forty years of age increased from seventy millions to eighty-seven millions, an increase of only 24.3 per cent. In other words the portion in which two-thirds of the cases of diabetes begin increased 2½ times as fast as the portion in which only one-third of the cases begins.

Moderate regular exercise helps the body to burn up sugar. Little physical effort is required now-a-days upon the part of large numbers of people whose ancestors were compelled to use their muscles vigorously in the daily routine of life.

Overnourishment plays a large part in the development of diabetes; 76.9 per cent. of Joslin's cases over twenty years of age were over standard weight by 11 per cent. or more at the beginning of the diabetes, while only 2.2 per cent. were less than standard weight by 11 per cent. or more. [1] The national consumption of sugar has risen enormously; but it is the fact that too many people consume too many calories in excess of their metabolic requirements which needs emphasizing rather than the exact articles of food consumed.

The increase in the proportion of Jews in our population, especially in our larger cities, accounts in part for the increased incidence of diabetes, as the incidence is especially high in this race.

The above mentioned factors probably have nearly reached their maximum effect in increasing the incidence of diabetes, and a further increase due to them is little to be feared. There is strong evidence to show that diabetes is a hereditary disease, but that it is a recessive rather than a dominant trait. This means that if two diabetics marry, one hundred per cent. of the children will be diabetic; that if a diabetic marries a person in whose family there is a diabetic tendency, fifty per cent. of the children will be diabetic; if two persons marry in each of whose families

1. Joslin, E. P.: TREATMENT OF DIABETES MELLITUS, Philadelphia, Lea & Febiger, 1928, page 157, table 68.

there is a diabetic tendency, twenty-five per cent. of the children will be diabetic; but if a diabetic marries a person with no familial diabetic tendency, none of the children will be diabetic. Before the advent of insulin, diabetics of reproductive ages seldom lived long enough to marry and beget children. Now it is a rather common event for a young adult diabetic to marry and produce children.

In view of the rapid increase in the incidence of diabetes, it is fortunate that it is the one common, serious disease for which we have an adequate therapeutic armamentarium. By modern management diabetes can now be so thoroughly controlled that we can truthfully tell our patients that we expect them to live just as long and be in just as good health as though they did not have diabetes, or as though they never had diabetes; provided irreparable damage has not already been done before it was brought under control. And yet it is not uncommon for insured persons to claim disability from diabetes, often stating that they have traveled to famous clinics at a distance from their homes and have been benefited temporarily, only to have a repetition of their disability shortly after returning home.

The modern management of diabetes is by diet and insulin. No medication by mouth has been proved to be capable of taking the place of insulin, in spite of the claims of numerous drug houses for their products to be taken by mouth. The principles of diet are few but exceedingly important. The diet may be described as simply a modification of the normal diet. It is not a highly restricted diet, nor are special diabetic foods necessary. The diet should contain enough calories to enable the patient to maintain his weight at a desired level and to carry on his usual activities with comfort. Overnourishment, undernourishment and hunger are all undesirable. The diet is divided into the proper amount for each patient of protein, fat and carbohydrate. The carbohydrate allowance is now sufficient so that most food-stuffs can be used. The important point is that the intake of protein, fat and carbohydrate must be constant each day, and the patients must be taught how this is to be accomplished. To a new patient this often seems difficult at first; but it usually becomes easy with

practice and proper instructions. It is this essential training which is so often lacking.

If by diet alone the urine is not sugar free and the blood sugar within normal limits, insulin is necessary. The patient who cannot be satisfactorily controlled by diet and insulin is extremely rare; and yet it is not uncommon to receive a report from a physician that a patient or claimant is refractory or resistant to insulin. If by "resistant to insulin" is meant that the blood sugar level cannot be reduced by large doses of insulin in an uncomplicated case of diabetes mellitus, such cases must be extremely rare. I have never seen such a case. Large doses of insulin are required temporarily during the presence of serious complications, especially acidosis and severe infections, one hundred to three hundred units during the first twenty-four hours being the usual requirement and sometimes much more than this. One of our patients, presenting marked allergic phenomena, during twenty months of stay in the hospital in 1929 and 1930, received 146,559 units, an average of 240 units a day, and over a period of 3½ months over 500 units a day were required. She left the hospital taking no insulin at all, and is still living and well without insulin. [2] In uncomplicated diabetes, however, 60 to 100 units a day is a large requirement, and of our hospital patients only about eight per cent. are discharged taking doses of this size.

The term "resistant to insulin" is often applied incorrectly to a patient when his fasting blood sugar is high, sugar is in the urine and yet the patient is having hypoglycemic reactions. Assuming that the diet is constant, the reason for the above circumstances is to be found in the fact that the size and spacing of the insulin doses are wrong. It is our object to keep the blood sugar at all times during the twenty-four hours below the usual level of the renal threshold; but well above the level at which hypoglycemic reactions occur. The effect of a single dose of insulin is lost in most patients after about eight hours. When a patient takes insulin at supper time, its effect is over by 2 to 3 A.M., and his carbohydrate metabolism is dependent on insulin from his

2. Karr, W. G.; Scull, C. W.; and Petty, O. H.: INSULIN RESISTANCE AND SENSITIVITY, J. of Lab. and Cl. Med. 18, 12, 1203 (Sept.) 1932.

own pancreas from then until the breakfast dose of insulin. If he is a severe diabetic, it is likely that his own pancreas will be unable to deliver enough insulin to prevent a marked rise of blood sugar in the interval from 2 A.M. to the before-breakfast dose of insulin, regardless of the fact that no food has been taken in this interval. This is the so-called night rise of blood sugar. Certain individuals are especially sensitive to insulin, sensitive in the sense that their blood sugar drops very rapidly following a dose of insulin. In such individuals the effect of a dose is lost, however, sooner than in nonsensitive patients. These patients present very unstable blood sugars, and their insulin requirements must be gauged with great accuracy. They are particularly likely to show a marked night rise. It is quite common to find the blood sugar to be 60 or 70 milligrams per cent. at 9 P.M., and then 300 or 400 the following morning. This rise can be prevented only by using a night dose of insulin between mid-night and 3 A.M. The term "sensitive to insulin" is sometimes used in an allergic sense, meaning that local or general allergic phenomena result from the injection of insulin, and due either to foreign protein or to the insulin itself. Such phenomena of a mild grade are rather common, but of a grade severe enough to interfere seriously with the use of insulin, are exceedingly uncommon in our experience.

The process of discovering the proper insulin doses in conjunction with a calculated diet is called "standardizing the patient". After a patient has been standardized and sent home, he will need to be carefully followed. Marked changes in tolerance are apt to occur, and the insulin doses must be changed accordingly. Even when a patient has been accurately standardized it is impossible to foretell what his future insulin requirements will be. Usually, when an uncontrolled case is brought under control, the tolerance increases due to the fact that the islets, having previously been forced to work at maximum capacity, are given a rest and an opportunity to recover some of their lost function. The amount of exercise taken, the state of gastro-intestinal tract and endocrine glands, worry, fatigue, all have an effect on the amount of insulin which will be required. The pancreas itself

seems to vary without demonstrable cause. I now have a gentleman of seventy-four, who has been in bed in the hospital for thirty-eight months because of a complete right hemiplegia, whose insulin requirement has varied between thirty-three and seventy-four units daily, there being periods of gradually increasing and decreasing need. He has been on the same diet and has had no other complications, conditions from day to day and month to month being as constant as one could conceive.

Most diabetics, when they have been adequately studied and trained, should be able to carry on with their usual occupations, having check-ups on their condition at necessary intervals. In most cases more than moderate physical effort is inadvisable. In others the chance that insulin reactions may occur should preclude certain hazardous occupations.

In spite of the fact that diabetes may be absolutely controlled, more diabetics are dying than ever before. The following mortality studies are from the Philadelphia General Hospital where we have a special department for diabetes, in which all the diabetics of the hospital are concentrated, regardless of other complications, except cases of open tuberculosis, unmanageable psychopathic conditions and contagious diseases. The department has its own wards, diet kitchen, laboratories and out-patient department, patients in the latter consisting only of patients who have been discharged from the wards. The department uses about 5,000,000 units of insulin each year.

During the years 1931, 1932, and 1933 there were 1,362 patients admitted to these wards of which 241 died while in the wards. In Table I these deaths are grouped by age and by cause of death. It should be stated that in most of these cases the diabetes was badly out of control at the time of admission to the hospital, many cases had never received any adequate treatment, and many others did not even know they had diabetes. We have been interested in the various groups for the purpose of criticizing our management of the cases after they came under our care, a discussion of which does not come within the scope of this paper. We have also been interested in estimating how many of

TABLE I.

Age	Acidosis Present at Adm. (a) Uncomplicated	(b) Complicated	(c) Complication cause of death	Gangrene	Heart	Infections	Apoplexy	Chr. Neph.	Cancer	Tbc.	Misc.	Sex M.	Sex F.	Total
0–10		1										1	0	1
11–20	2		3			(2)				(1)		0	5	5
21–30	2											0	2	2
31–40	2	2	1		2	1		(1)			1	2	7	9
41–45	1	1	3	1		5(3)			2	1		7	7	14
46–50		1	4	6	8	4	2(1)		1	(1)	2(2)	8	20	28
51–55	2	1	4	8(2)	2	4(2)	4	1	3		1	11	19	30
56–60	1		4	8(1)	11(1)	9(2)	1		3		3	13	27	40
61–65	1	3	2	10	12(1)	5	3	4	3	2	3(1)	20	28	48
66–70		1		11	6	3	2	1	1	1		14	12	26
71–75			2	14(1)	6	6	1	3	(1)			11	21	32
76+				2	2						2	0	6	6
	11	10	23*	60(4)	49(2)	37(9)	13(1)	9(1)	13(1)	4(2)	12(3)	87	154	241

*The complications causing death in these 23 cases are distributed in the figures in parentheses.

these deaths could have been avoided if the diabetes had been kept under control from the very beginning of the disease.

It will be noted in the table that 190 of the 241 deaths are in four groups, acidosis, gangrene, heart and infections, 78.8 per cent. of the total. In the Naunyn Era 60 per cent. of diabetics used to die in coma. We still think of coma as being that form of diabetic death most directly dependent upon diabetes for its occurrence. All deaths from acidosis can be prevented if the diagnosis is made early and adequate treatment commenced. During the three years 120 cases of acidosis were admitted of which 44 died. Of these 44 there were 11 which had no other complication (column "a"). There were ten cases in each of which there were one or more complications sufficiently severe to precipitate the acidosis, some of which cases may have died of the complications, but died too soon for this to be determined (column "b"). There were 23 other cases in each of which the patient was out of acidosis during the 48 hours or more before death occurred, or in which the complication was obviously overwhelming and would have caused death with certainty regardless of the acidosis. These 23 deaths have been distributed in Table I in the parentheses. When a patient dies in diabetic coma without other complications, the presumption is that someone has been at fault, the patient himself, his family or physician, and the circumstances under which this presumption is not warranted are exceedingly few.

Sixty of our cases died from gangrene, sixty-four, if four cases from the acidosis group are included. Next to coma, gangrene is the type of death whose pathology is most intimately dependent upon the diabetes for its development, and next to a coma death a gangrene death is the most unnecessary. The fundamental pathology of diabetic gangrene is an arteriosclerosis of muscular arteries, involving the intima, which encroaches upon the lumen of the vessel. The arteries most likely to be extensively diseased are those of the lower extremities and the coronaries. The blood supply is gradually and progressively diminished until a point is reached where, if an adequate collateral circulation has not been established, the blood supply will not

be sufficient to maintain the vitality of the tissues but it will still be too great for mummification to ensue. In senile arteriosclerosis the pathology is chiefly in the media and the lumen is not seriously encroached upon until a breach occurs in the intima with a sudden thrombus formation which quickly shuts off all blood and leads to mummification. Senile gangrene is less serious than diabetic gangrene because it is a dry gangrene and infection is much less likely to occur, and because proximal to the lesion the blood supply is likely to be relatively good.

No attempt has been made in our table to separate those cases which began as gangrene and those which began as infection of a toe or foot. In either case the proper course of action to be followed depends upon sizing up several factors, the amount by which the blood supply is diminished, the severity of the infection, the structures involved, and the general condition of the patient. Except for the presence of acidosis, the prognosis of a case of gangrene should not be much affected by the severity of the diabetes, for under proper management the diabetes can nearly always be controlled.

Deaths from diabetic gangrene ought not to occur. In the first place if the diabetes is under control from the beginning of the disease it is probable that the characteristic diabetic arteriosclerosis will not develop. There is much interest at present in the relative effect on the development of arteriosclerosis of high fat-low carbohydrate and high carbohydrate-low fat diets. Whatever the relative merits of these two diets, the main essential is that the diabetes shall be kept under control. The case of diabetes which is most apt to develop severe arteriosclerosis is not the severe diabetic, but the comparatively mild one which has been out of control over a period of years. In the second place, even if irreparable damage to the arteries has occurred, gangrene will probably not occur if proper attention is given to the feet. Calluses, corns, ingrowing toe nails, and traumatic injuries need expert attention, and for this purpose we have a special foot clinic. Proper foot exercises, well fitting shoes and above all cleanliness are essential. Thirdly, if gangrene does occur, lives in most instances will not be lost if the seriousness of the situa-

tion is recognized early and the proper treatment, medical or surgical, as the case requires, is instituted at once.

Both from the medical and economic points of view, gangrene is the biggest problem in our department. Over a period of 18 months there were 43 patients admitted who had comparatively mild lesions on their feet, not requiring surgical intervention; and yet even these comparatively mild cases cost the department 1,461 hospital patient days.

So far we may say that probably 21 coma deaths and 64 gangrene deaths should not have occurred. What shall we say of the heart deaths? We know that the uncontrolled diabetic is prone to develop coronary sclerosis. How many of these patients developed cardiac pathology and died earlier by virtue of their diabetes it is impossible to estimate. Coronary occlusion is very frequent. There were 14 cases of coronary occlusion out of 51 heart deaths, and we probably failed to diagnose other cases. In my private practice heart deaths in diabetics out-number deaths from all other causes combined.

The fourth large group of causes of death in diabetics is infections, a detailed list of which is presented in Table II. Skin infections in diabetics are, of course, notoriously common. The incidence of urinary tract infections also seems to be high. But these infections, in my experience, do not occur more often than in normal individuals if the diabetes is kept under control. Whether infections such as pneumonia and acute appendicitis are more likely to occur in diabetics than in individuals comparable except for the absence of diabetes, I am unable to say. The important thing to realize in connection with infections in diabetics is that as soon as an infection occurs there is immediately a loss of tolerance and the diabetes becomes much more severe. Many a case of mild or well controlled diabetes is thrown into severe acidosis by the occurrence of an infection, if proper measures are not taken to guard against this.

Having considered the principal causes of death in diabetes and pointed out that many diabetic deaths are unnecessary, it is interesting to speculate on what the effect on diabetic vital statistics would be if the entire diabetic population of the United States

TABLE II

Infections Causing Death

Carbuncle	7
Cellulitis	4
Erysipelas	1
Infected ulcer	1
Suppurative pyelonephritis	8
Lobar pneumonia	5
Broncho-pneumonia	5
Influenza	1
Pulmonary abscess	1
Multiple abscesses with blood stream infection	3
Acute appendicitis	2
Acute cholecystitis	1
Suppurative pyosalpingitis	2
Acute bacterial endocarditis	1
Acute phlegmonous duodenitis	1
Suppurative arthritis	2
Phlebitis	1
	46

had its diabetes under control. The vital statistics of diabetes are important. Diabetes is not a reportable disease so that the usual method of estimating the number of diabetics in the country is to multiply the number reported as dying each year by the estimated average number of years which a diabetic lives after acquiring diabetes. I believe that the number of diabetics dying in this country each year is considerably higher than our vital statistics show for the following reasons:

1. There must be many cases of diabetes which are never diagnosed. A considerable proportion of our deaths occurred in individuals who did not know they had diabetes until admission to the hospital. Either the patient had never felt sick enough to consult a physician until the onset of his final illness; or a urinalysis had never been done; or the diabetes had been so mild as to escape detection until it was aggravated by the complicating

disease. If this can be so in Philadelphia, how much more must it be true over large areas of the country where hospital facilities are not so readily available and urinalyses are done less routinely?

2. Although diagnosed, many cases of diabetes may not appear on the death certificates. As already pointed out, the final illness very frequently consists of one or more pathological conditions, not apparently directly dependent upon the diabetes, and which by their severity make the diabetes seem to the attending physician to be unworthy of being recorded as a contributory cause of death. Or the patient may have had diabetes well controlled for years and when he finally dies at an advanced age of heart disease or apoplexy, for example, it may seem to the attending physician to be unfair to record diabetes as a contributory cause, especially if he realizes that such a death will be tabulated as a diabetic death. Nothing said here is intended to discourage recording diabetes as a contributory cause of death on the death certificate, for I believe that it is highly desirable to record it in every case when it is known to exist.

3. In our largest cities the diabetic death rate, as reported by the local health departments, is fairly uniform; but the rates from the medium sized and smaller cities show astonishing inequalities. A few comparisons of cities from the same geographical areas in different parts of the country are made in Table III. In each instance a city with a high rate is compared with one or more with a low rate. In comparing Boston with the surrounding cities, it would almost seem as though the diabetics of the surrounding cities, about to die, migrated to Boston; and, of course, in part this may be true. Striking differences are observed in comparing Fall River with New Bedford, New Haven with Hartford, Utica with Albany, Paterson with Newark, Columbus with Akron, and New Orleans with other southern cities. When the rates are tabulated by the Bureau of the Census, however, from copies of the original death certificates, and according to the rules of the Manual of Joint Causes of Death, the rates of many of the low rate cities show a marked increase. Many of our city and state health departments do not possess the personnel with training in the compilation of vital statistics and

TABLE III

Death Rates Per 100,000 Population From Diabetes

City	Furnished by Local Health Departments [1]		Tabulated by the Bureau of the Census [2]	
	1931	1932	1931	1932
Boston	29.5	32.7	30.2	32.3
Cambridge	16.6	20.1	15.8	20.2
Chelsea	5.8	9.0	13.0	21.7
Lynn	14.6	13.6	27.3	23.4
Medford	4.8	12.5	11.3	22.5
Newton	7.4	8.6	11.8	20.7
Quincy	14.7	9.1	14.7	17.4
Somerville	7.6	10.4	20.0	27.6
Fall River	33.1	33.0	33.0	34.7
New Bedford	9.0	21.7	30.2	35.5
New Haven	45.5	43.6	38.7	46.1
Hartford	23.9	17.1	28.1	35.9
Utica	44.8	42.6	46.9	44.0
Albany	26.3	13.0	41.1	44.2
Paterson	45.4	46.0	44.7	45.4
Newark	14.4	16.5	29.6	32.5
Columbus	32.7	31.1	31.8	31.5
Akron	8.8	7.2	13.7	17.4
New Orleans	25.9	27.2	25.5	30.0
Birmingham	15.6	14.4	15.2	14.5
Dallas	12.5	16.5	11.0	17.2
Houston	11.9	12.0	16.8	18.4
San Antonio	22.9	18.2	23.7	20.4
Memphis	17.4	20.2	20.1	24.3

1. Hoffman, F. L.: The Diabetes Record of 1932, THE SPECTATOR 131:12 (July 6) 1933.
2. Murphy, T. F.: Personal Communication.

with sufficient continuity of service. Under such circumstances most of the diabetic deaths, when diabetes appears as a contributory cause, will not be tabulated as diabetic deaths, as prescribed by the Manual of Joint Causes of Death. But even with this factor taken into account, there are many inequalities which cannot be accounted for by the factors known to influence the incidence of diabetes, such as age distribution, habits, racial distribution, etc.

4. Even when the rules of the Manual of Joint Causes of Death are followed, there is a considerable percentage of deaths of diabetics attributed to other causes. All coma and gangrene deaths are, of course, tabulated as diabetic deaths. Diabetes, though it appears on the death certificate only as a contributory cause, takes precedence over cardiac and vascular diseases appearing as the principal cause. However, cancer and tuberculosis take precedence over diabetes. The deaths in the infection and miscellaneous groups are especially difficult to tabulate as being due to diabetes or to the complication. I submitted a list of 15 of our deaths from the various groups to the Bureau of the Census, the data being only what appeared on the death certificate, the results of which appear in Table IV. Of the 15 cases, ten are representative cases from our infection and miscellaneous groups (cases 6-15). Six of these are tabulated as not being due to diabetes. Out of our 241 deaths there are probably more than 40 which would not be tabulated as due to diabetes; just how many would depend on the individual judgment of the person making this tabulation.

It would be highly desirable to know accurately the incidence of diabetes in the United States and the relative weights of the factors known to be causing its increase. In using vital statistics in the study of these problems, faulty conclusions will be reached unless the above facts are borne in mind. It is submitted that the above considerations account in large part for the marked discrepancies in the vital statistics of diabetes as reported from different cities, from the various states the rates for which vary from Rhode Island with 26.9 to Arkansas with 8.3, and from urban as compared with rural populations.

TABLE IV

Sex	Age	Period Attended	Cause of Death	Duration	TABULATED AS DUE TO DIABETES "Yes" or "No" by the Bureau of the Census
1. F	74	6/28– 7/23	(a) Principal cause of death: Chronic Myocarditis with congestive failure (b) Contributory cause of death: Diabetes Mellitus Autopsy: No	6 weeks 13 years	Yes
2. F	50	10/14–12/12	(a) Coronary Occlusion (b) Diabetes Mellitus Autopsy: No Diagnosis confirmed by EKG	16 days 5 years	Yes
3. F	52	2/26– 2/27	(a) Cerebral Hemorrhage (b) Diabetes Mellitus Autopsy: Yes	18 hours Unknown	Yes
4. F	60	9/8 – 9/22	(a) Carcinoma of Pylorus (b) Diabetes Mellitus Operation: Gastroenterostomy Autopsy: No	4 months 21 months	No
5. F	20	8/21– 9/25	(a) Pulmonary Tuberculosis (b) Diabetes Mellitus Autopsy: No	5 months 4 years	No
6. M	44	10/5 –10/12	(a) Appendiceal Abscess Peritonitis (b) Diabetes Mellitus Operation: Abscess drained Autopsy: Yes	3 weeks 2 weeks	No
7. M	45	7/10– 7/13	(a) Acute Bacterial Endocarditis (b) Diabetes Mellitus Autopsy: Yes	9 days Unknown	Yes

TABLE IV—Continued

		Period			TABULATED AS DUE TO DIABETES "Yes" or "No"

TABLE IV—Continued

Sex	Age	Period Attended	Cause of Death	Duration	TABULATED AS DUE TO DIABETES "Yes" or "No" by the Bureau of the Census
8. M	61	7 hours	(a) Cavernous Sinus Thrombosis following furuncle of nose (b) Diabetes Mellitus Autopsy: Yes	2 weeks Several years	Yes
9. M	45	11/5 –11/12	(a) Lobar Pneumonia (type III) (b) Diabetes Mellitus Autopsy: No	7 days Unknown	No
10. M	67	7/25– 9/25	(a) Suppurative Pyelonephritis (b) Diabetes Mellitus Autopsy: Yes	2 months 3 years	Yes
11. F	56	9/29–10/29	(a) Toxic Adenoma of Thyroid (b) Diabetes Mellitus Operation: Thyroidectomy Autopsy: No	Impossible to secure history	No
12. F	74	10/2 –10/11	(a) Mesenteric Thrombosis Generalized Peritonitis (b) Chronic Myocarditis with congestive failure (c) Diabetes Mellitus Autopsy: Yes	60 hours 6 weeks 10 years	No
13. F	64	12/14–12/17	(a) Strangulated Incisional Hernia (b) Diabetes Mellitus Operation: Herniorrhaphy Autopsy: Yes	3½ days 5 years	No
14. M	50	11/4 –11/5	(a) Massive Hemorrhage from Peptic Ulcer (b) Diabetes Mellitus Autopsy: No	Unknown 2 years	No
15. F	50	9 hours	(a) Acute Hemorrhagic Pancreatitis (b) Diabetes Mellitus Autopsy: Yes	Unknown 4 months	Yes

DR. DILLON—There is very little which I can add. To briefly summarize, I have tried in my paper to bring out three things. In the first place, by modern treatment, I feel the vast majority of diabetics can now be kept in just as good health as though they didn't have diabetes. If a diabetic claims to be disabled, I believe one of four things is the matter. In most cases he probably has something else seriously wrong with him, or else he is being improperly treated, or if he is being properly treated, he is not following his treatment, or else he is simply using diabetes as an excuse.

Now so far as our diabetic statistics are concerned, I have tried to point out that most diabetics, when they die, have some other serious disease which at least plays a part in their death, and because of this, it is very difficult oftentimes to assign the diabetes or the contributing cause as the cause of death. In most cases, the diabetes is tabulated as the cause of death. However, in the Manual of Joint Causes of Death, there are 157 conditions which take precedence over the diabetes. That is not as bad as it sounds, because a great many of those come under the head of tuberculosis and cancer and traumatic conditions. Nevertheless there are a considerable number of diabetics dying each year which diabetics are not tabulated as diabetic deaths. I sometimes wonder whether the mortality statistics of life insurance companies in cases where the proofs of death contain two or more causes of death may not be criticized as regards uniformity, because of failure to follow rules laid down in the Manual of Joint Causes of Death.

Now so far as the micro methods are concerned, I think those of us who are doing a large amount of work in connection with diabetes realize and recognize the criticism which Dr. Joslin made. However, I am afraid in the life insurance business we will have to use micro methods. I don't see how we can use venous punctures.

I often wonder whether the time will come when we can insure our diabetics. If cases could be diagnosed early and if they could be referred to Dr. Joslin and if they could be fol-

lowed by Dr. Joslin, most important of all, over a long period of years, I would be willing to bet a lot of them. I could present a considerable group whose longevity would be just as good as that of normal individuals. However, when it comes to such a group as I see at the Philadelphia General Hospital, of an entirely different economic status, I wouldn't like to bet a cent on most of them. It makes a very great difference what the economic status of the patient is, and I presented this paper to-day largely to point out that the diabetic in the world at large are not the diabetic who are being treated by Dr. Joslin.

DR. FRASER—This brings us to the point of adjournment for today.

SECOND DAY

MORNING SESSION

DR. FRASER—Gentlemen, I asked you to get here at 9:00 o'clock. You did exactly as I expected you to do and got here at 9:30. Well, that's all right with me. I am glad you are here at 9:30.

I would like to ask or request all of those who have not registered to please register before luncheon today.

The first paper on this morning's program is that of "The Significance of the Systolic Murmur" presented by Dr. R. Wesley Scott, of Cleveland, Clinical Professor of Medicine at the Western Reserve University and Director of Medicine at the Cleveland City Hospital. Dr. Scott.

DR. SCOTT—Mr. President and Gentlemen: (Read paper).

THE SIGNIFICANCE OF THE SYSTOLIC MURMUR

By Dr. R. Wesley Scott
City Hospital, Cleveland, Ohio

In this paper I deal with the significance of the systolic murmur from the viewpoint of the clinician, duly aware, I trust, of the difference between clinical medicine and insurance medicine. This difference may be illustrated in the interpretation of the systolic murmur. The clinician is concerned with the mechanism of adventitious cardiac sounds. He tries to differentiate an intra and extra cardiac systolic murmur, a functional and an organic murmur. He observes the associated evidences of myocardial damage in the hope of determining the individual patient's cardiac future. These questions may be of academic interest to the insurance medical director, but he has found that the most expert clinical opinion does not answer his question: What is the influence of a systolic murmur on the mortality of the group? This admittedly important problem I do not presume to answer. Its solution will not come from clinicians, but from mortality data accumulated over a period of years from large groups at various age levels. When all the facts are available, the clinician may help to explain them, and in so doing he may also find it necessary to change some of his opinions, particularly regarding the prognostic significance of systolic cardiac murmurs.

The attitude of the profession toward abnormal heart sounds has changed widely at different periods since Laënnec first used a roll of paper as a stethoscope in 1816. In the interval immediately following Laënnec's discovery, physicians observed that patients dying of heart failure frequently showed murmurs during life, and the conclusion was reached that murmurs were a sign of serious heart disease. As time went on clinical and pathological observations accumulated as well as knowledge of the heart sounds, particularly those from the different valve

areas. It became evident that murmurs were associated with certain architectural deformities in the heart valves. Physicians found that their clinical conceptions of the state of certain valves were often confirmed at the post-mortem table and for many years a cardiac murmur was regarded as a manifestation of valvular disease. On the other hand, some individuals exhibiting cardiac murmurs, particularly systolic murmurs, were found at post-mortem to have no demonstrable change in the valvular apparatus and hence a functional murmur came to be recognized. Clinicians generally, however, were so impressed with the ominous significance of cardiac murmurs as a manifestation of a diseased heart, that a clinical differentiation between a harmless and an organic murmur was rarely made. More often the individual with an unimportant systolic murmur was managed as a case of heart disease. This extreme view appeared to have dominated clinical teaching well up until the turn of the last century. For example in a well-known book on the heart published in 1898 we find this statement, "Of all the signs of cardiac disease murmurs are usually those most confided in". In other words, heart murmurs had come to be so intimately associated with a damaged heart in the minds of the majority of the profession, that the presence of a murmur alone was usually sufficient evidence to incriminate the heart. The time was ripe for a swing of the pendulum of thought away from such an extreme view, but instead of taking up a more nearly mid-position, in many quarters at least, the pendulum has gone to the opposite extreme in the past twenty-five years particularly with regard to the significance of systolic cardiac murmurs. Impressed with the frequency of systolic murmurs in the sound heart of young people who were observed to have no evidence of myocardial insufficiency, and who survived a normal expectancy, some prominent clinicians recently have gone so far as to say that systolic murmurs have no significance whatever; that no one ever dies of mitral insufficiency; that the stethoscope has retarded rather than advanced our knowledge of heart disease. Such views clearly represent the opposite extreme to that held by physicians of a past generation. The truth of the matter

appears to lie somewhere in the middle ground. It is from this mid-position, as it were, that I propose to consider the significance of the systolic murmur heard over the precordium.

It is not my purpose to present a detailed account of the various factors producing systolic murmurs, but the causes will be briefly discussed and certain observations bearing on the interpretation of systolic murmurs will be given here. Vibrations imparted to the walls of the heart and great vessels by the circulating blood cause audible sounds which vary in intensity with the amplitude of the vibration. In other words, the more rigid and thick the wall the less intense the sound. Vibrations of the walls of the heart and vessels of sufficient amplitude to cause murmurs occur when the fluid particles of blood no longer flow in parallel stream lines, but in tortuous paths or eddies. The motion now becomes turbulent.

The commonest cause of "turbulence" in the blood stream is an increase in velocity flow. Thus it is, that in conditions producing an increase in the minute volume output of the heart beyond the so-called "critical velocity" a murmur may be heard. Since the greatest velocity occurs during systole, we find that the vast majority of such murmurs are systolic in time. This explains the appearance of a systolic murmur in a sound heart following some brisk exercise or in individuals with a simple tachycardia from emotional excitement. Systolic murmurs from increased minute volume output are also seen in patients with hyperthyroidism and in individuals with fever.

A second common cause of turbulent motion with eddy formation results from the passage of blood through a narrow orifice directly into a channel of greater caliber. Thus, incompetent mitral valves which do not seal the orifice, permit a regurgitant stream of blood into the left auricle during ventricular systole and we hear a systolic apical murmur. Another familiar example is the basal systolic murmur caused by passage of blood from the left ventricle through a narrowed aortic orifice.

A third and less common cause of turbulence in the blood stream occurs usually in diseased hearts, in which a band or cord of tissue is so disposed as to vibrate in the blood column. This

may happen in cases of ruptured papillary muscles or chordae tendineae; again in instances of syphilis of the aortic valve, in which a leaflet may be ruptured and vibrate in the regurgitant stream into the left ventricle much as a sail vibrates in a breeze.

We have seen that blood flow beyond the so-called "critical velocity" may cause murmurs in normal hearts. Since the viscosity of blood is a factor in velocity (as viscosity decreases the velocity increases) we find systolic murmurs in a variety of anemic states. Here another factor may be considered, namely, that of cardiac dilatation, so often associated with high grade anemias. The mitral ring, anatomically a part of the left ventricle, may be widened as the ventricle dilates and a normal mitral valve may not entirely close the orifice, or the papillary muscles relaxed by disease may permit an eversion of the mitral valve. In either case a relative mitral insufficiency results with the production of an apical systolic murmur.

In some people extraneous factors are present which modify murmurs as well as heart sounds. An increase in the anterior posterior diameter of the thorax, chronic pulmonary emphysema of the large lung variety, and obesity, all render murmurs less audible than in thin-chested individuals or in children.

It is usually, although not always, possible to distinguish between an intra and extra cardiac systolic murmur. The latter are most often the so-called cardio-respiratory murmurs resulting from the movement of air in the lung tissue adjacent to the heart. Such murmurs usually possess a respiratory quality, are not widely transmitted, vary with different positions of the body and with different phases of the respiratory cycle, often disappearing with a deep inspiration.

Interpretation of Systolic Murmurs

I come now to the most difficult part of my subject and in discussing it I have tried to base my remarks as much as possible upon observations that have come within my own experience both clinically and at the post-mortem table. This method of handling the subject is open to the criticism that one's personal experience is necessarily limited, that views based thereon must

suffer accordingly, but I know of no better approach to the subject before a group such as yours.

The insurance applicant presenting obvious signs and symptoms of organic heart disease with or without a systolic murmur, presents no difficulty to the insurance examiner. For example, individuals with hypertension and a systolic murmur due to a relative mitral insufficiency from dilatation of the left ventricle are not an insurance risk. The real problem, both to the physician and the insurance examiner, is the individual in whom a systolic murmur is the only apparent manifestation of present or future heart disease. This is the type of case with which we are chiefly concerned here.

Apical Systolic Murmurs

We may define this murmur heard at the apex of the heart as blowing in character, beginning at or shortly after the first heart sound, and continuing for an appreciable length of time during systole. The intensity of the murmur and the proximity of the stethoscope to the heart determine the extent to which it may be transmitted away from the apex. Loud murmurs may be accompanied by a palpable thrill. Generally speaking the louder the murmur the farther it is transmitted. Here the question may be asked: "Is the transmission or non-transmission of the adventitious sound of any value in the diagnosis?" Is an apical systolic murmur transmitted to the left axilla, to the base of the heart, or even over the left chest posteriorly, more likely to be a manifestation of heart disease than the soft systolic blow sharply localized to the region of the apex? I think this question can be answered in the affirmative. Certainly more often than not such murmurs are associated with some form of cardiovascular disease and are due to a regurgitation of blood from ventricle to auricle through an incompetent mitral valve. We must of course, remember that the loudness and transmission of a murmur do not necessarily increase as the heart disease advances. For instance, a slight mitral leak attended by a vigorous ventricular contraction may produce a loud murmur. As the mitral aperture widens and the ventricle dilates, the murmur may fade or

entirely disappear. It is not always possible clinically to determine the cause of the mitral leak, but the post-mortem examination often tells the story The valves may be scarred and the chordae tendineae shortened from an old inflammatory process such as rheumatism, or, in elderly people they may be more or less rigid from fibrosis and calcification. In either case they are unable to seal the mitral orifice during ventricular systole. More commonly a dilated left ventricle widens the mitral ring or displaces the papillary muscle thus shortening the chordae tendineae so that even normal valves may not close the mitral orifice.

We may now consider a group of cases in which an apical systolic murmur is the sole evidence of a scarred rheumatic mitral valve. I refer to those people who give no history of a previous rheumatic infection, who have had no clinical evidence of myocardial insufficiency, and appear healthy at the time of examination. The following brief case history is a fairly typical example.

In 1919 a boy, then aged 15 years, was found to have an apical systolic murmur not transmitted to the axilla. There was no history of rheumatic infection, no demonstrable enlargement of the heart fluoroscopically, and no evidence of myocardial impairment functionally. A normal cardiac response to effort was found. The murmur was regarded as insignificant and the lad was allowed to carry on as a normal boy. In college he engaged in competitive athletics with no apparent difficulty. After graduation, at age 24, the apical systolic murmur still present was interpreted as a functional murmur and he was accepted as a standard insurance risk. Five years later at age 29 he developed symptoms of a low-grade generalized infection. After six weeks a splenic infarction occurred and the blood culture contained streptococcus viridans. Other signs of sub-acute bacterial endocarditis appeared and the patient died five months after the onset of the disease. At no time in the clinical course was there any evidence of myocardial insufficiency. At post-mortem the heart weighed 350 grams. The mitral valve was scarred from a previous rheumatic infection, but it was not narrowed to the point of stenosis. On it and extending up over the mural endocardium

of the left auricle were vegetations typical of sub-acute bacterial endocarditis. Here then was an individual whose early rheumatic infection was so mild that it passed unnoticed. As is often the case in childhood, the heart was affected. An early mitral valvulitis underwent healing, but the resulting architectural distortion in the valves rendered them incompetent. However, the resulting organic mitral incompetency caused no apparent myocardial insufficiency. Similarly the histological changes found at post-mortem were of no apparent significance. For at least fourteen years prior to the patient's death (not from heart failure but from infection) the systolic apical murmur afforded the only clue to the diagnosis of organic heart disease.

The interpretation of an apical systolic murmur in the group illustrated by the above case report is little more than the expression of a clinical opinion in the individual case, but in arriving at this opinion the informed clinician to-day is more influenced than his predecessors of a generation ago by certain facts concerning rheumatic heart disease. These, so far as they bear on our present problem, may be briefly summarized as follows: A definite percentage (variously estimated between twenty and twenty-five per cent.) of people with rheumatic hearts are unaware of a previous attack of rheumatic infection. A questionable organic mitral insufficiency therefore cannot be eliminated solely on the basis of a negative history of rheumatism. A rheumatic infection occurring at that most dangerous age (8 to 16) affects the heart in a varying degree in a high percentage of cases (estimated from 75 to 85 per cent.). The valve most frequently damaged is the mitral and the only sign of this damage in some cases may be a soft systolic blow at the apex. After five or more years some damaged mitral valves go on to stenosis and such individuals usually develop evidence of progressive myocardial impairment and die of heart failure. Others remain stationary and exhibit only mitral insufficiency—certainly clinically—and the future danger in these patients is not congestive heart failure, but sub-acute bacterial endocarditis. Some people with rheumatic mitral insufficiency, of course, escape this fatal infection and survive a normal expectancy, but it is my opinion that on the

whole they are poor insurance risks. It would be interesting to know how many people, particularly in the third decade of life with an apical systolic murmur, accepted as standard risks, later die of sub-acute bacterial endocarditis. It seems likely that mortality in this group may be a considerable factor in the disparity between actual and anticipated expectancy in persons with systolic apical murmurs.

We have seen that a diseased mitral valve due to rheumatism may in the course of several years become narrowed with the development of the clinical signs of mitral stenosis. In such cases there may be an interval of several years before the signs of stenosis appear during which the only manifestation of heart disease may be an apical systolic murmur.

Ten years ago I saw a physician, then aged 28, who eight years previously at age 20 had had a mild rheumatic fever. There were no subsequent attacks and he had no symptoms referable to the heart following the initial attack. A systolic apical murmur not well transmitted to the axilla was heard, the heart was not demonstrably enlarged fluoroscopically, and the electro-cardiogram was normal. The murmur was interpreted as a functional one and he was accordingly accepted as a standard insurance risk. Five years later examination revealed the same murmur a little louder than previously. Acceleration of the heart, however, with exercise or amyl nitrite revealed a diastolic rumble and snappy first sound of mitral stenosis. In other words, it was not until thirteen years after the initial rheumatic infection that the signs of mitral stenosis appeared and these were elicited only after acceleration of the heart. Now, eighteen years after the initial infection, auscultatory signs of mitral stenosis are heard with a palpable diastolic thrill, the heart shows a typical configuration of mitral stenosis fluoroscopically, and the physician himself, to-day aged 38, is quite aware of his limitations in cardiac reserve. His case is illustrative of another group of individuals with rheumatic heart disease in whom an apical systolic murmur for years may be the only manifestation of organic mitral insufficiency.

Only a few years ago a prominent American clinician and teacher, in discussing the diagnosis of mitral regurgitation, said:

"Physicians diagnose mitral regurgitation when they hear a loud apical systolic murmur which is extremely common in all sorts of non-cardiac disease as well as in health"; and again, "a diagnosis of mitral regurgitation without stenosis is never justified". It is, of course, well known that an apical systolic murmur may be heard over a sound heart under a variety of conditions particularly in young people, but if influenced by such extreme views as those we have just quoted, the examiner will interpret many apical systolic murmurs as of no consequence, when, as a matter of fact, they may signify an organic mitral insufficiency from rheumatism. As we have seen, one such group succumbs to the fatal infection usually due to the streptococcus viridans, the other after a period of years develops mitral stenosis and dies of heart failure.

Pertinent to this discussion is the question of the age at which most people with mitral insufficiency become insurance problems. We have seen that rheumatism affects the heart most frequently between the ages of 8 and 16, so that early in the third decade of life we may expect the evidence of a damaged mitral valve to appear in the majority of cases. From this time to well within the fourth decade they may be problems in diagnosis, but their number diminishes rapidly after forty, a dangerous age for the rheumatic heart. At this time of life many are either dead or are permanent cardiac cripples; others have such clear evidence of heart disease that they are no longer an insurance problem.

In discussing age groups mention may be made here of the apical systolic murmur which appears more often in men than in women, at or beyond forty years of age. A structural defect in the mitral valve due to sclerosis may be present; more often, however, we deal with a relative incompetence of the mitral orifice from left ventricular dilatation. This condition commonly associated with hypertension is of little concern to the insurance examiner, but in people with no elevation of blood pressure an accurate estimate of the systolic murmur may be important in directing our attention to the possibility of coronary disease. Recently I examined a fifty-two year old business executive of sedentary habits who desired to increase his insurance by a con-

siderable amount. As late as five years ago he was accepted for a large policy. The only evidence of heart disease either subjective or objective elicited in the ordinary clinical examination was an apical systolic murmur, transmitted to the left axilla, but not to the base or to the left posteriorly. It seemed likely that this murmur had appeared some time within the past five years as no record of it was found in previous insurance examinations. There was no elevation in the blood pressure and the accessible arteries were not unduly sclerosed. Fluoroscopically, the aorta was dense, definitely widened and the left ventricle demonstrably enlarged, a finding not elicited by percussion because of the configuration of the thorax. The electro-cardiogram afforded indisputable evidence of myocardial damage. Not all cases of hypertensive heart disease or coronary disease with or without angina pectoris have an apical systolic murmur, but its appearance in a man beyond forty, particularly if it is known that no murmur existed previously, should make us think of organic heart disease. Not all such murmurs will be correctly evaluated, but it is safer, I believe, to regard them with suspicion, at least until reliable evidence to the contrary is forthcoming.

Finally I need only mention the systolic murmur heard at the apex, but transmitted from the base. Lesions of the aortic orifice, such as aortic stenosis, may cause a loud systolic murmur heard at the apex, but usually the origin of such a murmur is not difficult to determine.

Systolic Murmurs at the Base of the Heart

We come now to a consideration of systolic murmurs heard at the base of the heart. Since the significance of these basal murmurs varies widely, depending on their origin at the aortic or pulmonary area, every effort to localize them should be made. It is my opinion that aortic systolic murmurs are so often associated with organic disease in adults, that from an insurance standpoint it is dangerous to regard them in any other light. A common error is made in ascribing a systolic murmur heard over the aortic area, to transmission from the pulmonic area.

The pathological changes responsible for aortic systolic murmurs involve the root of the aorta, the aortic ring and the valve cusps. The first part of the aorta may be widened from syphilis or arterio-sclerosis with or without hypertension. The vessel in this situation may be greatly distorted from sclerosis and here I may mention an interesting fact, namely, that some definite relation exists between longevity and sclerotic changes in the first few centimeters of the aorta. In individuals who survive the proverbial three score and ten years, the first four or five centimeters of the aorta are singularly free of arteriosclerotic changes, whereas, if this part of the vessel is seriously involved the individual does not see seventy years. It may be that some factor responsible for the localization of the sclerotic process at the root of the aorta also determines in some way the state of the coronary arteries in elderly people. Whatever the explanation, one has no difficulty in proving the relation between longevity and the state of the first part of the aorta at the post-mortem table.

The aortic valve cusp may be distorted from rheumatism or some other chronic inflammatory process so as to cause an obstruction from the left ventricle into the aorta with the production of an aortic systolic murmur. Again, sclerotic changes in the sinuses of Valsalva may extend to the aortic cusps which become more or less rigid, project into the lumen of the aorta during systole and cause the systolic murmur of aortic stenosis. This murmur is usually harsh and when loud is accompanied by a palpable systolic thrill. In some instances the thrill is only elicited by having the patient lean forward and expire as deeply as possible bringing the palpating hand as near as possible to the aortic valve area. The systolic murmurs originating in the aorta are usually followed by a second heart sound which is frequently not heard after murmurs due to valve stenosis.

Pulmonic Systolic Murmurs

We come now to the commonest of all heart murmurs, the systolic murmur heard over the pulmonic area. It is usually blowing in character, fairly sharply localized, does not obliterate

the first heart sound and is often followed by an accentuated second heart sound. It is so common in healthy young people during a forced expiration, particularly in the dorsal position, that under these circumstances it may be regarded as physiological. The mechanism of its production is not known, but a dilatation of the pulmonary artery from increased pressure during forced expiration, the proximity of conus to the chest wall in young people, a constriction of the vessel from change in position, are all factors which may be concerned.

Less common, but much more significant, is the pulmonary systolic murmur due to pathological changes in the pulmonary artery associated often with hypertrophy and dilatation of the right ventricle in instances of mitral stenosis and chronic pulmonary emphysema. Congenital heart disease, pulmonary stenosis with or without architectural defect, patent ductus arteriosus, often but not always cause a systolic murmur over the pulmonary area. In these cases the diagnosis rarely rests solely on an interpretation of the murmur. In other words, the pulmonary systolic murmur of pathological significance is so frequently accompanied by other clear evidence of disease that its interpretation should not be a serious matter to the insurance examiner.

Summary and Conclusion

Excepting physiological systolic murmurs originating over the pulmonic area and extra cardiac respiratory murmurs all apical and aortic systolic murmurs in adults merit consideration. The faint ones are usually but not invariably benign; louder ones are frequently associated with some type of cardio-vascular disease.

An apical systolic murmur for years may afford the only clinical evidence of a damaged rheumatic mitral valve which may go on to stenosis or become the seat of the usually fatal sub-acute bacterial endocarditis. Aortic systolic murmurs are more often than not a sign of organic disease. The current clinical teaching that tends to disregard the significance of all systolic cardiac murmurs is just as unsound as the older view which

associated every adventitious cardiac sound with organic disease. Somewhere between these two extremes lies the truth and it is from this mid-position that I think we should attempt an evaluation of systolic murmurs.

DR. FRASER—The first discusser of this paper is Dr. Edward J. Campbell, Medical Director of the New York Life Insurance Company. Dr. Campbell.

DR. CAMPBELL—Mr. President and Gentlemen: (Read paper.)

When we were requested to undertake this discussion we knew only the title of Dr. Scott's paper. We planned of course to use the material from a mortality study upon which we were working. When Dr. Scott's completed paper was compared with the results of our study, we were delighted to find that our results both supplemented and substantiated his opinions. Since this is true, it may be that the difference of opinion between clinical medicine and insurance medicine, is due to a different approach to the problem, but in estimating prognosis there is no great difference of opinion if Dr. Scott's proper and common sense suggestion is followed—namely—viewing the problem from a mid position between the extremes.

Since our work is primarily that of selecting risks, we have been impressed by the fact that in our selection we frequently take into consideration factors which we do not and can not code for actuarial purposes and hence are not available in a study based on actuarial cards alone. The primary purpose of our study was to determine not only an up-to-date summary of the Company's experience, but also to discover whether or not those factors which we considered significant actually did have an effect on the mortality experience.

We chose to begin with the substandard issues of January 1, 1925, to December 31, 1931, carried to the policy anniversary in 1933. The date of January 1, 1925, was selected because on this date a new method of classifying heart murmurs became effective and on this date we began to utilize new standards of heart ratings which were unchanged throughout the period studied.

Our method was determined only after discussion with all members of our Medical Board who assisted in the study. We realize that our subdivisions and classifications are somewhat arbitrary and may not meet with universal approval. Some clinicians, particularly, may be critical of our methods but it must be remembered that most clinical reports are compiled from the records of specialists in heart disease who have the benefit of a complete history given with their patients' full cooperation. Our material is based upon the reports of a large group of average general practitioners whose histories are sometimes sketchy and incomplete and to whom applicants for insurance are not always relating their complete history.

In this discussion we use the term "mitral regurgitation—rheumatic group" to designate all systolic murmurs at the apex, both with and without transmission, having a history of rheumatic fever, tonsilitis, chorea, scarlet fever or other streptococcic infections; "simple mitral regurgitation", to refer to systolic murmurs at the apex, transmitted to the axilla or back and also systolic murmurs at the apex not transmitted, provided hypertrophy was also reported. While this discussion deals only with systolic murmurs at the apex, all substandard cases having a heart murmur of any type were studied. In order to secure complete details it was necessary to go back to the original files. These files were reviewed by the members of our Medical Board who transcribed upon work sheets all pertinent facts in regard to each individual case. After all cases were transcribed upon our work sheets, we then undertook to classify simple mitral regurgitation into homogeneous groups for study. Table I is an exact reproduction of the rules we followed. The same rules were used for mitral regurgitation—rheumatic group, except for the necessary change in reference to a history of rheumatic fever. However, our original subdivision into five groups was found to be impractical for the purpose of a mortality study and we therefore made three classifications which are as follows:

CLASS A—is comprised of simple heart murmurs with no unfavorable history from any source and no other significant impairment. (Group I and II, Table I)

CLASS B—simple heart murmurs plus an unfavorable history, usually of a cardiovascular-renal type, from a source other than our own application papers and not confirmed by our examiner. (Group III, Table I)

CLASS C—simple heart murmurs plus one or more additional impairment of considerable significance, making it really a group of complex impairments. (Group IV, Table I)

We will call your attention particularly to Class B, which is a group we believe to have significant impairments not available in the usual study from actuarial cards alone. Class C, of course, contains those which the actuarial cards show have complex impairments, but also contains an appreciable percentage of complex impairments which ordinarily would not have been secured from the cards alone.

Table II shows that of 7,302 entrants with all degrees of hypertrophy, only 60% or 4,337 can be described accurately as simple mitral regurgitation; that of the 346 dead only 40.5%, or 140, can be classified as simple mitral regurgitation. While according to our classification, only 60% of the entrants are simple mitral regurgitation and 40% have other impairments, this is of no significance unless our classification also shows a deviation in the mortality experience, and this it does show.

CLASS A—192%
CLASS B—298%
CLASS C—408%

In mitral regurgitation—rheumatic group the figures are quite parallel. Simple murmurs plus rheumatic history are 65% of the total and contribute 56% of the total dead. The mortality ratios are:

CLASS A—337%
CLASS B—374%
CLASS C—723%

In considering the insurability of simple mitral regurgitation, Table II indicates that in only three of the nine groups studied does the mortality experience justify consideration as insurance risks, even on a substandard basis. They are the ones in which it would be expected—namely:

1—CLASS A without hypertrophy
2—CLASS A with slight hypertrophy
3—CLASS B without hypertrophy

In mitral regurgitation—rheumatic group, only the most carefully selected risks, namely Class A without hypertrophy, yield an experience indicating that they can be considered insurable.

We have noted the inconsistency in the mortality ratio of simple mitral regurgitation Class B with slight and moderate hypertrophy. The results are even higher than similar material in the rheumatic group. The only explanation we have to offer is that on going back to the cause of death, we find 10 deaths from accident and 13 from pneumonia, while no other similar group had more than 3 deaths from accident and 2 from pneumonia. In addition, this group having hypertrophy, are more likely to be those in which a rheumatic history has been concealed. Certainly it is a group in which a high mortality is not surprising, since they all have an organic murmur, hypertrophy and a history of at least one other impairment.

In considering our study by policy year Table III shows the highest mortality in Class A of simple mitral regurgitation in the first two years and there is a very definite improvement with duration. In Class B material improvement is evident only after five years of duration. In contrast, in the mitral regurgitation—rheumatic group in Class A, the mortality increases with duration.

Table IV very forcibly supports Dr. Scott's opinion that organic systolic murmurs are more significant at the younger ages. Taking Class A we have the following figures:

Ages	Simple Mitral Regurgitation	Mitral Regurgitation—Rheumatic Group
15–39	271%	469%
40 and over	144%	185%

Even when subdivided into smaller age groups, in every class the relative mortality is highest at the younger ages.

In distribution of deaths by cause, as expected, cardiovascular-renal deaths predominate. (Table V) Organic heart disease, myocarditis, endocarditis, embolism and thrombosis contribute the greater number. One result we can not explain—the percentage of cardiovascular-renal deaths in Class A without hypertrophy is greater than with hypertrophy. (Table VI) This is

not true of the rheumatic group. There were several deaths from suicide due to ill health and in each case these individuals had seriously impaired hearts. There were several deaths from pneumonia that we suspected were actually heart deaths with a terminal pneumonia, but this could not be definitely established. It is noticeable in our tabulation that deaths attributed to syphilis of the heart and aorta were conspicuous by their absence—only one death in the entire series. In clinical experience syphilis is one of the major causes of heart disease. We would suggest that our cases are those handled by the average practitioner who may have failed to establish the correct etiology, or who, in deference to his client's family, refrained from making a public record of syphilis, as would be necessary in properly completing the death certificate. It is interesting to note in Table V the mathematical precision of the ratio of actual to expected dead from cardiovascular-renal disease. There is an increase in all groups but one, the progression taking place in the following order: 6, 7½, 13, 13½, 12 and 26 times. Dr. Scott inquires in regard to the deaths due to sub-acute bacterial endocarditis. In simple mitral regurgitation there were 16 deaths specified as from this cause, or 5% of the total deaths. In mitral regurgitation—rheumatic group there were 20 deaths, or 9% of the total. Several others were given as endocarditis, chronic endocarditis or bacterial endocarditis which may also have been subacute bacterial endocarditis.

In considering systolic murmurs from a pathological point of view, Class A is the only group in which there is surely no other factor affecting the mortality. In comparing Class A of simple mitral regurgitation with the Medical Impairment Study, we have the following figures:

Hypertrophy	New York Life 1925-1931	Medical Impairment Study 1909-1927
None	169%	224%
Slight	185%	234%
Moderate	370%	476%
Total	192%	240%

We believe these figures clearly establish that when the material is carefully revised by the Medical Board the results are quite different from a study made from actuarial cards alone. Carry-

ing this idea a step further we combined Class A and Class B for comparison with the Medical Impairment Study. Class B, as you know, differs from Class A only in having an unfavorable history from sources other than our own application papers, and which was not confirmed on examination when we accepted the risk. In the usual study from Actuarial cards alone, Class A and Class B probably would fall together—therefore we believe these classes combined closely approximate the material in the Medical Impairment Study. Table VII shows that these classes combined yield a result on the whole more favorable than the Medical Impairment Study and the difference is particularly striking in simple mitral regurgitation without hypertrophy. In the mitral regurgitation—rheumatic group, however, our experience is more favorable only in those without hypertrophy and definitely higher when slight and moderate degrees of hypertrophy are combined. Carrying our idea still one step further, we combined Classes A, B and C for comparison with the Medical Impairment Study. Table VIII shows the New York Life experience at all ages 280%—the Medical Impairment Study 240%. Obviously our higher figures are due to the inclusion of Classes B and C and we believe indicates the probability that the figures of the Medical Impairment Study are higher than our Class A because similar material was included in that study. We believe that if the material for the Medical Impairment Study had been subdivided as we subdivided our material, the results would be similar.

Further comparing the New York Life experience with the Medical Impairment Study, both studies show the highest mortality below age 40. The inclusion of Class C probably causes the New York Life higher figures of relative mortality. They may equally be due to the inclusion of a greater portion of data with hypertrophy. (Table VII) Nevertheless, the results are quite parallel. The New York Life experience is considerably higher at the 1 to 5 years duration and lower at the 6 to 10 years duration. The mitral regurgitation—rheumatic group by ages shows a startling difference below and above age 40. Dr. Scott has expressed the opinion that rheumatic heart disease

most frequently has its onset at a relatively young age. By the third decade many are dead and many of the survivors are cardiac cripples. When we accept individuals suffering from rheumatic heart disease—say between the ages of 20 and 30, we know there still remains several years of potential danger for each individual and this is directly reflected in the higher mortality below age 40. Entrants at age 40 and up represent the survivors with a minimum amount of damage and with the history of rheumatic fever remote—hence the more favorable experience.

Four mortality studies by this Company are compared with the Medical Impairment Study in Table IX. The figures for issues of 1919 to 1923 and the Medical Impairment Study are very similar, while the New York Life issues of 1914 to 1918 and 1925 to 1931 are similar and both of the latter were made from the original files which had been reviewed and classified by our Medical Board. These issues, nearly a decade apart, studied in a similar manner, indicate that there is little change in our experience with mitral regurgitation without hypertrophy. It is a further indication that a study made from mortality cards alone produces results different from a similar study of the same material when revised and classified by the Medical Board.

We believed it advisable to investigate carefully the difference betwen simple mitral regurgitation and mitral regurgitation with a history of rheumatic fever. We felt that if the cases were carefully selected the best of each group might yield a somewhat similar mortality experience. This did not prove to be true. Reference to Table II shows that simple mitral regurgitation without hypertrophy, Class A, had a mortality experience of 169%, whereas Class A of mitral regurgitation without hypertrophy—rheumatic group, had a mortality experience of 220%. Furthermore, a review of all of the simple mitral regurgitations reported dead, totalling 347 policies, showed that in only 23 cases (7%) was there information on the death certificate or from any source indicating that the lesion was actually due to rheumatic heart disease. It may be that when an attack of rheumatism is so mild or so far in the past as to be forgotten, it is of less significance. This is indicated by the fact that the majority of studies show an

improvement in mortality with duration and the best relative mortality experience at the older ages.

Dr. Scott states that "the insurance problem is the individual in whom a systolic murmur is the only apparent manifestation of present or future heart disease" and it is exactly this group in which our figures indicate the most favorable mortality experience. Some clinicians contend that many systolic murmurs at the apex, even though transmitted, may be functional, accidental or extra-cardiac—and this may be true. However, as Dr. Scott states, "it is not always possible clinically to determine the cause of a mitral murmur". This fact, together with the estimation that from 20 to 25% of the individuals suffering from rheumatic heart disease are unaware of a previous attack of rheumatism, would indicate that the systolic murmurs actually of an innocuous character are far outweighed by those ostensibly innocuous, but in fact of serious significance. The studies of Rogers and Hunter include approximately 23,000 policies, with 1,160 deaths. Our present study includes approximately 14,000 policies with 811 deaths. These, together with the Medical Impairment Study of 45,025 policies, with 2,430 deaths, are figures which are conclusive and cannot be disregarded. They indicate that systolic murmurs with transmission or with hypertrophy or both, as a group, yield a mortality experience well above normal and certainly from an insurance point of view can not be considered functional in character and of no significance.

There is a very important point brought out by Dr. Scott, to which we can well give consideration in making our selection. A mitral leak with a vigorous contraction results in a loud murmur, but later this same heart with a dilated ventricle may reveal a faint murmur, not transmitted, or even no murmur at all. Are we justified in disregarding a history of organic heart disease simply because no murmur is found on examination? Are we justified in disregarding a long record of even a simple mitral regurgitation just because a current examination reveals no murmur? A study of Class B strongly indicates that unfavorable cardiovascular-renal history from any source, when coupled with a murmur, cannot be disregarded and it is reasonable to believe

that a similar history, even without a murmur, may be significant.

While the Company's experience with this entire group of murmurs on the whole has been satisfactory, in view of the extra mortality provided for, it is quite obvious that Class C and a certain portion of Class B are not insurable on the basis of the rating we are now accepting. It would appear that other companies have selected their risks in a manner similar to our selection, since the Medical Impairment Study, which contained material contributed by several companies, yields an experience very similar to ours when all our material was studied together. It is not at all improbable that we as selectors have been affected by the swing of a pendulum to a liberal point of view and that medical propaganda in this direction, together with previous reports of a favorable mortality experience, probably influenced us in the selection of complex impairments.

Rogers and Hunter in 1924 stated as follows: "It seems probable from this study that the relative effect of the heart lesion decreases with the duration of the policy and that it is more serious at the younger than at the older ages at entry". The present study confirms this statement. Our findings do not support the opinion that there is an increasing mortality in these groups of heart murmurs, but we believe they do indicate the necessary care in selection in order to secure a satisfactory experience. As would be expected, the carefully selected group of simple mitral regurgitation gives the most favorable results. When simple mitral regurgitation is combined with slight hypertrophy, the group is still insurable, but individuals having a murmur plus more than one other unfavorable factor, form a group that is not insurable.

We have not emphasized the mitral regurgitation—rheumatic group, as we believe the tables are self-explanatory. The results fairly parallel the results of the simple mitral regurgitation group. Our study confirms previous reports and opinions that a history of rheumatic fever is of considerable significance and definitely predicts a mortality experience considerably higher than that of simple mitral regurgitation.

The chief conclusions of Dr. Scott's paper are that systolic heart murmurs are significant at all ages—at the younger ages because often indicative of a heart damaged by rheumatic infection,—at the older ages often indicative of a sclerotic heart, hypertensive heart disease or syphilis. We believe our study and previous mortality studies fully substantiate Dr. Scott's conclusions.

I wish to emphasize that I am only the spokesman in reporting this study. It was made possible only by the assistance of nine other members of our Medical Board who spent many laborious hours compiling our work sheets from the original files. Chief Medical Director Fraser was our guiding spirit and chief adviser. Dr. Harold E. Bogart of our staff contributed more time, energy and helpful suggestions than any other one individual. Dr. Hunter, Chief Actuary, contributed valuable advice and the full cooperation of his department. Assistant Actuary Walter G. Bowerman compiled Tables II to IX from our work sheets. His advice and cooperation have been invaluable.

TABLE I

GROUP I—Simple Heart Murmurs (Mitral Regurgitation)

(1) Those having no adverse history or other findings and no unfavorable report from other sources.
(2) Those having an unfavorable report, from other sources, of murmur the same or less degree.
(3) Blood Pressure on examination
Systolic 142 or lower but not below 100.
Diastolic 96 or lower.
Pulse pressure 69 or less.

GROUP II—Heart Murmurs (Mitral Regurgitation) with Borderline Impairments on Examination

(1) Albumin—any amount—unless clearly accidental and only one finding.
(2) Casts—any amount—unless clearly accidental and only one finding.
(3) Sugar—any amount—unless clearly accidental and only one finding.
(4) Any test meal, dextrose test or blood sugar test.
(5) Blood Pressure
Systolic over 142 or below 100 } Averages
Diastolic over 96 } Averages
Pulse pressure over 69 } Averages
One finding systolic 160 or more.
One finding diastolic 100 or more

TABLE I—Continued

(6) Pulse
Rapid—90 up. Two or more findings and only one normal finding.
Irregular—of any degree, even one finding, and even if later not found on same application.
(7) Arteriosclerosis—any degree.
(8) Any history or other finding which, according to a strict interpretation of our then current rules, called for an added rating of +15 or more.

GROUP III—Heart Murmurs (Mitral Regurgitation) with Borderline Records from Other Sources

(1) Albumin—any amount—unless clearly accidental and only one finding.
(2) Casts—any amount—unless clearly accidental and only one finding.
(3) Sugar—any amount—unless clearly accidental and only one finding.
(4) Any test meal, dextrose test or blood sugar test.
(5) Blood Pressure
Systolic over 142 or below 100
Diastolic over 96
Pulse pressure over 69
} Averages
One finding systolic 160 or more.
One finding diastolic 100 or more.
(6) Pulse
Rapid—90 up. Two or more findings and only one subsequent normal finding.
Irregular—of any degree, even one finding, and even if later not found on same application.
(7) Arteriosclerosis—any degree.
(8) Any history or other finding which, according to a strict interpretation of our then current rules, called for an added rating of +15 or more.
(9) Any heart murmur more serious.
(10) Any report of hypertrophy of greater degree than reported on examination.
(11) Any report of rheumatism or other streptococcic infection (chorea, tonsilitis, endocarditis, etc.), not investigated or cleared up on our papers.

Group IV—Heart Murmurs (Mitral Regurgitation) with one or more other impairments calling for a rating of +30 up and/or any cardiovascular-renal impairment calling for a rating of +15 up. Basic rating for build or tuberculosis in family over 125.

Group V—Heart Murmurs Other Than Mitral Regurgitation

It is well to note here that we included in the mitral regurgitation group all cases of a systolic murmur at the apex, not transmitted, in which there was reported hypertrophy of the heart. The murmurs coded systolic, functional, at the apex, without transmission, with a definite history of rheumatic fever or other infection, were placed in the mitral regurgitation—rheumatic group.

TABLE II

Substandard Issues of 1925 to 1931, inclusive—Carried to Anniversary in 1933. Men and Women. Expected Dead by Company's Standard.

MITRAL REGURGITATION

Group	Hypertrophy	Entrants	Number Dead	Ratio	Expected Dead	Mortality Ratio
Class A	None	2,745	80	2.9%	47.35	169%
	Slight	1,173	35	3.0	18.92	185
	Moderate	419	25	6.0	6.76	370
Total		4,337	140	3.2%	73.03	192%
Class B	None	1,232	45	3.7%	26.09	172%
	Slight	375	41	10.9	6.48	633
	Moderate	175	20	11.4	3.02	662
Total		1,782	106	5.9%	35.59	298%
Class C	None	766	61	8.0%	16.29	374%
	Slight	289	25	8.7	5.67	441
	Moderate	128	14	10.9	2.53	553
Total		1,183	*100	8.5%	24.49	408%

MITRAL REGURGITATION—RHEUMATIC GROUP

Group	Hypertrophy	Entrants	Number Dead	Ratio	Expected Dead	Mortality Ratio
Class A	None	1,113	33	3.0%	14.97	220%
	Slight	718	32	4.5	9.71	330
	Moderate	321	33	10.3	4.37	755
Total		2,152	98	4.6%	29.05	337%
Class B	None	596	30	5.0%	9.74	308%
	Slight	244	15	6.1	3.45	435
	Moderate	107	10	9.3	1.51	662
Total		947	55	5.8%	14.70	374%
Class C	None	120	11	9.2%	2.01	547%
	Slight	54	7	13.0	.71	986
	Moderate	35	5	14.3	.46	1087
Total		209	23	11.0%	3.18	723%

NOTE: Those with no hypertrophy were by actual tabulation and so was the total including all degrees of hypertrophy. The sum of expected dead data for slight and moderate hypertrophy was obtained by subtraction. They were then subdivided into the proportion which actual deaths bore to all entrants.

*One death with large amount of hypertrophy is omitted here from Class C of the mitral regurgitation group; thus the mortality ratio is 408% instead of 412% as in Table III.

TABLE III

Substandard Issues of 1925 to 1931, inclusive—Carried to Anniversary in 1933. Men and Women. Expected Dead by Company's Standard.

MITRAL REGURGITATION

By Policy Year

	CLASS A			CLASS B			CLASS C		
Policy Year	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
1– 2	55	25.63	215%	42	12.32	341%	30	8.47	354%
3– 5	61	32.44	188	52	16.21	321	53	11.13	476
6–10	24	14.96	160	12	7.06	170	18	4.89	368
Total	140	73.03	192%	106	35.59	298%	101	24.49	412%

MITRAL REGURGITATION—RHEUMATIC GROUP

By Policy Year

	CLASS A			CLASS B			CLASS C		
Policy Year	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
1– 2	34	10.56	322%	23	5.54	415%	11	1.17	940%
3– 5	43	12.93	333	21	6.53	322	8	1.44	556
6–10	21	5.56	378	11	2.63	418	4	.57	702
Total	98	29.05	337%	55	14.70	374%	23	3.18	723%

TABLE IV

Substandard Issues of 1925 to 1931, inclusive—Carried to Anniversary in 1933. Men and Women. Expected Dead by Company's Standard. All degrees of Hypertrophy are included.

MITRAL REGURGITATION

By Age at Issue

Age at Issue	CLASS A			CLASS B			CLASS C		
	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
15–29	41	12.80	320%	19	3.54	537%	17	2.50	680%
30–39	33	14.54	227	20	6.57	304	19	4.50	422
40–49	46	23.49	196	40	11.93	335	25	7.25	345
50 and over	20	22.20	90	27	13.55	199	40	10.24	391
Total	140	73.03	192%	106	35.59	298%	101	24.49	412%
15–39	74	27.34	271%	39	10.11	386%	36	7.00	514%
40 and over	66	45.69	144	67	25.48	263	65	17.49	372
Total	140	73.03	192%	106	35.59	298%	101	24.49	412%

MITRAL REGURGITATION—RHEUMATIC GROUP

By Age at Issue

Age at Issue	CLASS A			CLASS B			CLASS C		
	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
15–29	37	7.88	470%	16	2.55	627%	6	.71	845%
30–39	36	7.67	469	18	4.00	450	6	.70	857
40–49	18	9.11	198	7	4.15	169	7	1.02	686
50 and over	7	4.39	159	14	4.00	350	4	.75	533
Total	98	29.05	337%	55	14.70	374%	23	3.18	723%
15–39	73	15.55	469%	34	6.55	519%	12	1.41	851%
40 and over	25	13.50	185	21	8.15	258	11	1.77	621
Total	98	29.05	337%	55	14.70	374%	23	3.18	723%

TABLE V

Substandard Issues of 1925 to 1931, inclusive—Carried to Anniversary in 1933. Men and Women. Expected Dead by Company's Standard.

MITRAL REGURGITATION

Cause of Death	CLASS A			CLASS B			CLASS C		
	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio
Organic Heart Disease	35	5.60		21	2.97		32	2.03	
Myo- and Endocarditis	34	.89		26	.45		14	.31	
Angina pectoris	2	1.48		2	.81		3	.55	
Embolism and Thrombosis	10	.10		6	.04		10	.03	
Cerebral Hemorrhage	1	2.75		0	1.53		4	1.03	
Arterio-sclerosis	3	1.10		2	.59		1	.40	
Nephritis and Bright's Disease	7	2.96		3	1.53		6	1.04	
Total	92	14.88	618%	60	7.92	758%	70	5.39	1299%
			6x			7½x			13x

MITRAL REGURGITATION—RHEUMATIC GROUP

Cause of Death	CLASS A			CLASS B			CLASS C		
	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio
Organic Heart Disease	21	1.94		17	1.08		8	.24	
Myo- and Endocarditis	36	.33		16	.18		5	.03	
Angina pectoris	3	.49		0	.28		1	.05	
Embolism and Thrombosis	3	.02		0	.01		1	.00	
Cerebral Hemorrhage	4	.88		0	.51		0	.12	
Arterio-sclerosis	2	.37		1	.20		0	.04	
Nephritis and Bright's Disease	1	1.08		1	.57		1	.13	
Total	70	5.11	1370%	35	2.83	1237%	16	.61	2623%
			13½x			12x			26x

TABLE VI

Substandard Issues of 1925 to 1931, inclusive—Carried to Anniversary in 1933. Men and Women. Expected Dead by Company's Standard.

MITRAL REGURGITATION

Distribution of Deaths by Cause

	CLASS A		CLASS B		CLASS C	
	$\bar{s}$ Hyp.	$\bar{c}$ Hyp.	$\bar{s}$ Hyp.	$\bar{c}$ Hyp.	$\bar{s}$ Hyp.	$\bar{c}$ Hyp.
Total Dead.....	80	60	45	61	61	39
C.V.R. Dead.....	53	39	28	32	43	27
(x) % C.V.R.....	66	65	62	52	70	69
Diff.	−1		−10		−1	

MITRAL REGURGITATION—RHEUMATIC GROUP

Distribution of Deaths by Cause

	CLASS A		CLASS B		CLASS C	
	$\bar{s}$ Hyp.	$\bar{c}$ Hyp.	$\bar{s}$ Hyp.	$\bar{c}$ Hyp.	$\bar{s}$ Hyp.	$\bar{c}$ Hyp.
Total Dead.....	33	65	30	25	11	12
C.V.R. Dead.....	16	54	15	20	7	9
(z) % C.V.R.....	48	83	50	80	64	75
Diff.	+35		+30		+11	
(x)—(z)	+18	−18	+12	−28	+6	−6

NOTE: Under Rheumatic Group the proportion of C.V.R. deaths to total is distinctly greater under cases with hypertrophy than under those without hypertrophy. Under Mitral Regurgitation there is a slight tendency the other way.

It is a curious circumstance that for cases without hypertrophy the C.V.R. proportion is greater under Mitral Regurgitation than Mitral Regurgitation —Rheumatic Group; the exact reverse being true for cases with hypertrophy.

TABLE VII

Comparison of New York Life Study of 1925 to 1931 with Medical Impairment Study of 1909 to 1927. Men and Women—All Ages— By Degree of Hypertrophy. Expected Dead by Company's Standard.

MITRAL REGURGITATION

Class A and B	Hypertrophy	New York Life Substandard Issues of 1925 to 1931, inclusive — Carried to Anniversary in 1933			M.I.S. Substandard Issues of 1909 to 1927, Inclusive—Carried to 1928		
		Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
	None	125	73.44	170%	1,231	549.08	224%
	Slight	76	25.40	299	394	168.45	234
	Moderate	45	9.78	460	196	41.20	476
	Slight and Moderate combined	121	35.18	344	590	209.65	281
Total		246	108.62	226%	1,821	758.73	240%

MITRAL REGURGITATION—RHEUMATIC GROUP

Class A and B	Hypertrophy	New York Life Substandard Issues of 1925 to 1931, inclusive — Carried to Anniversary in 1933			M.I.S. Substandard Issues of 1909 to 1927, Inclusive—Carried to 1928		
		Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
	None	63	24.71	255%	410	147.23	278%
	Slight	47	13.16	357			
	Moderate	43	5.88	731			
	Slight and Moderate combined	90	19.04	473	192	56.95	337
Total		153	43.75	350%	602	204.18	295%

TABLE VIII

Comparison of all Mitral Regurgitations of New York Life Study of 1925 to 1931 with all Mitral Regurgitations of Medical Impairment Study of 1909 to 1927. Expected Dead by Company's Standard.

MITRAL REGURGITATION

	N. Y. L.			M. I. S.		
	By Age at Issue			By Age at Issue		
	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
15–39	149	47.64	313%	1,094	422.76	259%
40 and over	198	76.16	260%	727	335.97	216%
All ages	347	123.80	280%	1,821	758.73	240%
	By Policy Year			By Policy Year		
1–5	293	98.77	297%	1,113	464.75	239%
6–10	54	25.03	216%	545	226.26	241%
Total	347	123.80	280%	1,658	691.01	240%

MITRAL REGURGITATION—RHEUMATIC GROUP

	N. Y. L.			M. I. S.		
	By Age at Issue			By Age at Issue		
	Actual Dead	Expected Dead	Mortality Ratio	Actual Dead	Expected Dead	Mortality Ratio
15–39	119	24.19	472%	421	128.45	328%
40 and over	57	19.46	293%	188	81.66	230%
All ages	176	43.65	403%	609	210.11	290%
	By Policy Year			By Policy Year		
1–5	140	35.50	394%	360	126.87	284%
6–10	36	8.15	442%	160	58.86	272%
Total	176	43.65	403%	520	185.73	280%

TABLE IX

Comparison of four New York Life Studies with Medical Impairment Study. Expected deaths have in each case been expressed in terms of the companies' standard for the given period of experience.

MITRAL REGURGITATION—WITHOUT HYPERTROPHY

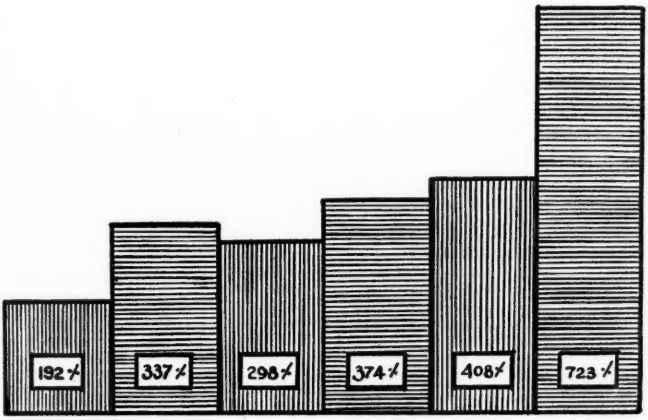
Substandard Policies

Years of Issues	Year Published	Actual Dead	Expected Dead	Mortality Ratio
New York Life Rogers and Hunter 1896–1922	1924	805	427.40	188%
New York Life Rogers and Hunter 1914–1918	Medically Revised 1924	134	85.00	158%
New York Life Rogers and Hunter 1919–1923	1924	145	63.99	227%
Intercompany "Medical Impairment Study" 1909–1927	1931	1,231	549.08	224%
New York Life Present Data 1925–1931	Medically Revised 1934	125	73.44	170%

NEW YORK LIFE INSURANCE COMPANY

MORTALITY RATIOS SIMPLE Vs. RHEUMATIC
ALL DEGREES OF HYPERTROPHY

TABLE II



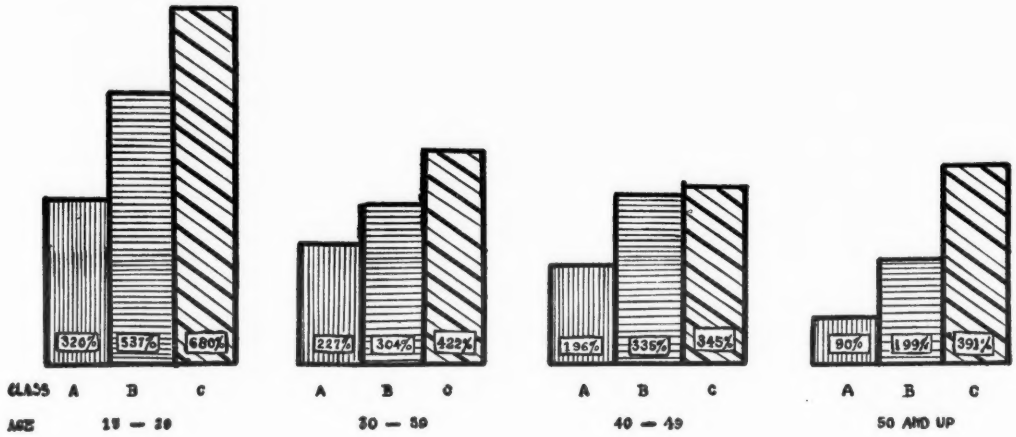
CLASS A CLASS B CLASS C

MITRAL REGURGITATION

NEW YORK LIFE INSURANCE COMPANY

Simple Mitral Regurgitation Mortality Ratios by Age Groups

TABLE IV

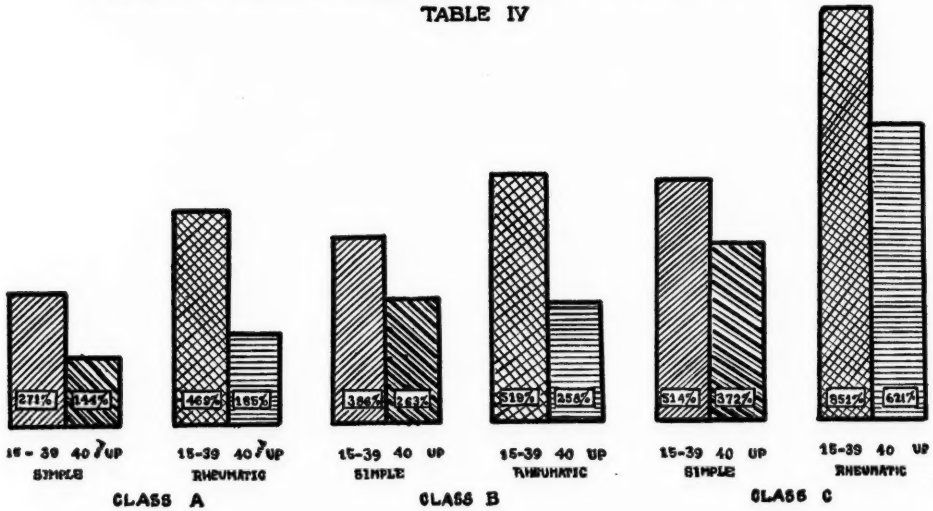


NEW YORK LIFE INSURANCE COMPANY

Mortality Ratios By Age Groups

Simple Mitral Regurgitation vs. Rheumatic

TABLE IV



NEW YORK LIFE INSURANCE COMPANY

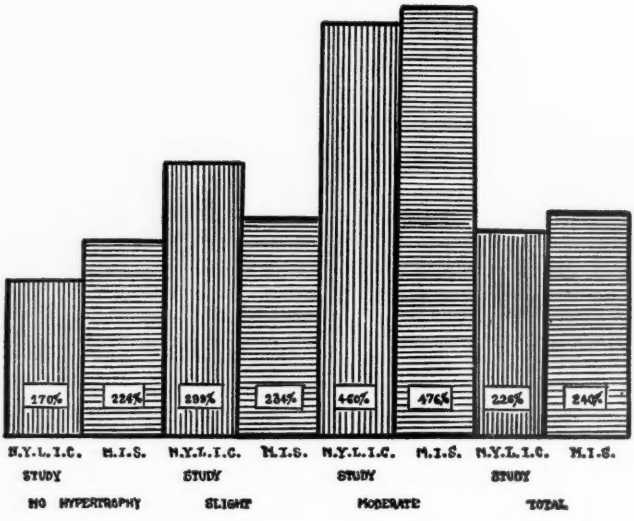
Simple Mitral Regurgitation

Comparison of New York Life Study with M.I.S.

Class A and B N.Y.L.I.C. Compared with M.I.S.

Substandard Issues

TABLE VII

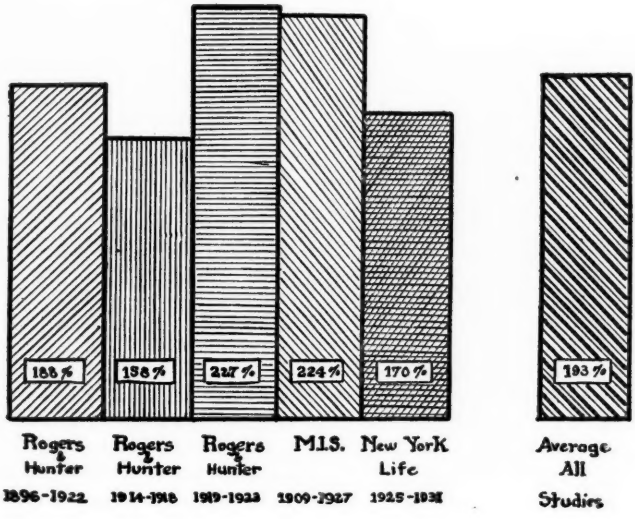


NEW YORK LIFE INSURANCE COMPANY

Comparative Mortality Ratios

Mitral Regurgitation — without Hypertrophy

Table IX



DR. FRASER—In the program you will note the name of Dr. Henry F. Starr, Medical Director of the Pilot Life Insurance Company, as the second discusser. Unfortunately, Dr. Starr was taken with a very severe illness and was unable to complete his paper. I was in considerable perturbation in regard to a second discusser, so I spoke to Mr. Hunter about the matter, and he, with his usual kindness and interest in the organization, contributes the following discussion of this paper:

MR. HUNTER—The Association is fortunate in having a paper on this subject from a physician of such breadth of view and wide experience as Dr. Scott. I hesitate to discuss it, but I am doing so at the request of your President. It has been frequently stated that the companies must base their decisions on the examinations made by all types of physicians and not only on those by specialists like Dr. Scott; accordingly there may be differences between his results and the experience of the life insurance companies.

The first mortality investigation of functional heart murmurs was published by Dr. Oscar H. Rogers and myself 15 years ago. While the material was not large it indicated that there was little extra mortality at the younger ages whereas such murmurs were of significance at the older ages. The striking feature was that the death rate from heart disease among men above 35 years of age was four times the normal. Stimulated by that investigation eleven of the insurance companies prepared their material for a joint study which showed that the relative mortality increased with attained age. Again in 1924 the experience of the New York Life was published under cases treated as substandard from 1896 to 1922. It showed a mortality practically normal at the younger ages but 35% extra at ages 40 and over; the deaths from heart disease were 22% of the total. The term "functional murmur" seems to have been superseded by a more precise description of the condition. In the recent "Medical Impairment Study" there were three groups which would apparently come under the definition of functional, and in these classes the extra mortality was of greater importance

at the older ages. The layman naturally wonders why the relative mortality in the case of the so-called functional murmurs apparently increases with advancing age. Is it not due to including in this group many cases which are really organic?

Dr. Scott's reference to heart murmurs with a history of rheumatism is very enlightening. In my young days it was customary to decline all cases of heart murmurs with a history of "rheumatic fever". It was also the general practice to rate severely applicants with a history of rheumatism, although showing no evidence of heart trouble. In the book entitled "The Medical Adviser in Life Assurance" (1882), the author, Dr. E. H. Sieverking, stated that the large majority of heart disease in Great Britain was due to rheumatic fever or acute rheumatic arthritis, that this malady rarely leaves the sufferer unscathed and that cases are common where the cardiac lesion can be traced later in life to rheumatic fever.

The recent investigations of our Joint Committee have shown clearly that the physicians of the last generation had good reason for their opinion with regard to the importance of heart murmurs following rheumatism, although they had little statistical evidence to support them. The experience of the American and Canadian companies indicates that a constant systolic apex murmur transmitted to the left with a history of rheumatism calls for a provision of from three to four times the normal mortality. The result is that few companies are accepting applicants on any terms with an organic murmur if a history of rheumatism has occurred within recent years.

Dr. Scott states that from 20% to 25% of persons with a rheumatic heart do not have knowledge of a previous rheumatic infection. When we add to this number those who conveniently forget such a history when examined for insurance, it is obvious that the mortality of the group without a history of rheumatic infection is affected by the inclusion in the group of many who actually had a rheumatic history which was not revealed at the medical examination. Both clinicians and insurance companies agree that the mortality from heart disease with rheumatic his-

tory is high in early life. May this be due to the rheumatic infection damaging the heart muscles as well as the valve to a greater degree than other infections?

Dr. Scott states that the apical systolic murmur appears more often in men than in women. Is this due to the different conditions under which men live than women or may it be the result of the former taking more opportunities to determine their physical condition than the latter? It may also be due to hesitation on the part of women to consult a man about their condition. We notice, for example, in our non-medical business that the proportion of women compared with men is twice as high as where medical examination is required.

In the paper the question is raised "Is an apical systolic murmur transmitted to the left axilla, to the base of the heart, or even over the left chest posterior, more likely to be a manifestation of heart disease than the soft systolic blow sharply localized in the region of the apex?" In the experience of the companies published in 1931 there was one class of apex murmur, systolic constant, transmitted to the left, and another, the same type but not transmitted. If these represent, in general, the two classes which Dr. Scott has in mind then his opinion is correct from the statistical standpoint, namely, that the applicants with the former have a distinctly higher mortality than those with the latter.

Dr. Scott's statement that "the current clinical teaching that tends to disregard the significance of all systolic cardiac murmurs * * * is * * unsound" is undoubtedly correct and is proved by the experience of our companies. It is unfortunate, however, that there is not more cooperation between the clinical and actuarial points of view. Few specialists have either the time or the training to read the results of the insurance companies' investigations. In the present instance it is most satisfactory to note that Dr. Scott, in dealing principally with individuals, comes in large measure to the same conclusions as the life insurance companies when considering groups of persons.

DR. FRASER—Dr. Scott will complete the discussion and I am sorry that I was unable to let him read this paper before he had an opportunity to make a further discussion.

DR. SCOTT—I don't know that there is very much more to discuss. It is naturally very gratifying to me to find that the experience of you insurance men agrees very largely with the opinions I have tried to present to you here this morning based upon clinical and pathological studies of a large number of heart cases. It is obvious that I could deal with this subject only from the standpoint of the clinician who has kept in close touch with the post mortem table, and from this experience it has seemed to me, as I indicated clearly in the paper, that somewhere in the middle ground lies the truth in the consideration of the significance of systolic heart murmurs.

I want to take this opportunity to thank you very much for the place on the program which Dr. Fraser has given me.

DR. FRASER—The next subject on the program will be the paper, "Coronary Sclerosis as an Insurance Problem" by Dr. Arlie R. Barnes. I regret, as I told you yesterday, that Dr. Barnes is not able to be with us. A call took him away three or four days ago and he is now on the high seas. I have asked Dr. Benjamin M. Baker, Jr., one of the discussers of the paper and a member of the Cardio-vascular Laboratory of Johns Hopkins University, to read Dr. Barnes' paper in his place. Dr. Baker.

DR. BAKER—(Read Dr. Barnes' paper.)

CORONARY SCLEROSIS AS AN INSURANCE PROBLEM

By Arlie R. Barnes, M. D.
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Examiners and adjusters for insurance companies have stated to me that they felt the medical profession was unfair to them at times when adjusting questions related to coronary sclerosis. The inference of that statement is that the physician is more interested in protecting his patient than he is in doing justice to the insurance company. It is my belief that, so far as insurance problems connected with coronary sclerosis are concerned, such disagreements as arise between physicians and insurance officials are due to a lack of a common understanding of the fundamental facts about coronary disease. In the interest of a better understanding I want to present some of these fundamental concepts as I see them.

It is a fashion to state that coronary sclerosis is on the increase, particularly since 1920 or 1921. This apparent increase is subject to many qualifications. Levy and his coworkers have called attention to the phenomenal increase in frequency of the clinical diagnosis of disease of the coronary arteries since Herrick's paper appeared in 1919, and they remind us that physicians have become not only "heart minded" but "coronary conscious". The most dependable data relating to this question are to be obtained from studies at necropsy, in consecutive years, of degrees of coronary sclerosis. It is essential that the same pathologist or pathologists, actuated by the same concepts, should determine the grading of coronary sclerosis throughout the period. Willius, Smith, and Sprague conducted such studies over a period of a decade. Although fluctuations in the degree of coronary sclerosis occurred year by year, no definite trend could be

demonstrated in the plotted curves, and they concluded that "at least a span of twenty years is required in order to answer the question whether diseases of this type are increasing in incidence."

Levy, Bruenn and Kurtz have made a similar study of consecutive cases coming to necropsy in the years 1910 to 1931, inclusive. The incidence of coronary disease during these years shows a gentle but perceptible rise, although from 1919 to 1931 this increase is almost imperceptible. These workers indicated that this slight increase obtains at all ages. Bearing in mind the tremendous impetus that interest in this subject has received in the past fifteen years, one wonders to what degree the pathologist has been influenced by this condition. Similar pathologic studies of the trend of coronary sclerosis covering the decades from 1920 to 1940 will carry much more conviction.

Numerous judicial and experienced medical observers are convinced that angina pectoris and acute coronary occlusion are on the increase. It is conceivable and even probable that angina pectoris, exclusive of acute coronary occlusion, has increased without any change in the incidence and degree of coronary sclerosis as a result of the increasing stress under which people live. It is noteworthy that the anginal syndrome often appears in people at a time when they have been subject to unusual mental stress. Statistics of mortality indicate that death from heart disease is on the increase. This situation is mitigated, however, by many facts, and the increase is much more apparent than real. In the first place there is not the slightest doubt that the physician's index of suspicion and ability to recognize the existence of heart disease due to coronary sclerosis and hypertension has increased tremendously since 1919. Due to the conquest of infectious diseases in early life, there has been a gradual preservation of the population to the age when degenerative heart disease must take its toll. Dublin and Armstrong express this by stating that, in a generation, "the population of the country has increased by 62 per

cent., whereas the population at ages past 45 has more than doubled." Riesman and Harris regard this as a triumph of preventive medicine. The Bouldans showed that, in 1900, 27.5 per cent. of the population of New York City were in the age group of from thirty-five to sixty-four years as compared to 33.8 per cent. in 1930. They further contended that the registered increase in cardiac deaths in that area has occurred because fewer deaths have been ascribed to apoplexy, Bright's disease, and to senility, and they pointed out that there has been no significant increase in the death rates from cardio-arterio-renal disease in thirty years.

Dublin and Armstrong's tables disclose that, among white men, more than fifty-five years of age and among white women more than sixty-five years of age, deaths from heart disease have been increasing in the last decade and that they have attained a level appreciably above that of the years preceding the influenza epidemic. It is obvious that almost all deaths from heart disease at those ages are due to disease of the heart muscle, the result of coronary sclerosis or of hypertension, or combinations of the two. Admitting the more frequent identification of coronary disease by physicians and the changes in classification of the cause of death, these observers are convinced, nevertheless, that death from disease of the heart muscle is definitely on the increase, although statistical studies indicating an increase may give an exaggerated estimate of its degree.

Coronary sclerosis manifests itself in a variety of ways the commonest of which is in the syndrome of angina pectoris. In the absence of pain the condition may indicate its presence by the occurrence of paroxysmal nocturnal dyspnea. The clinical picture of progressive myocardial failure, in the absence of angina pectoris or of hypertension, may have its basis in coronary sclerosis. An attack of acute coronary occlusion may occur without any antecedent evidence that the heart was diseased. Some patients who have suffered from such an attack may be remarkably free of cardiac symptoms after recovery from the acute attack. Finally, there is

a group of patients who during life never have had signs or symptoms of coronary sclerosis but at death are found to have an advanced degree of coronary disease. Willius has designated this unrecognizable condition as "occult coronary sclerosis".

The diagnosis of angina pectoris continues to offer difficulty. Mistakes are of two kinds: First, failure to recognize the syndrome, and second, interpretation of thoracic pain as angina pectoris when it is not. There are three characteristics of the anginal syndrome which constitute a diagnostic triad of the condition: 1. The distress is situated behind the sternum. The pain in angina pectoris may be felt elsewhere in the thorax, in the neck, jaws, or in the epigastrium, but if such pain is to be designated angina pectoris, then the relationship of the symptoms must conform in all other respects to that characteristic of the disease. The term "precordial pain" is highly objectionable, inasmuch as pain so described usually is in the apical region or in the left axillary line and such pain rarely proves to be the result of coronary sclerosis. If distress on effort has its situation behind the sternum, the presumption is strong that coronary disease is its cause; if the pain is situated in the left anterior or lateral thoracic wall, then a diagnosis of angina pectoris can be made only on the basis of features characteristic in all other respects. 2. A characteristic feature of angina pectoris is that the attack is precipitated by any factor that increases cardiac work. These factors are, in order of importance, physical exertion, particularly walking, excitement, ingestion of food, or combination of these. This essential relationship of cardiac overload to the onset of an attack is diagnostic only if the seizure stops in a few moments after the provocative factor ceases to operate. 3. The attack of angina pectoris is brief. The typical attack is over in a few minutes, seldom lasting longer than fifteen minutes. Attacks of substernal pain lasting half an hour or longer always should lead to the suspicion that coronary occlusion has occurred.

It cannot be emphasized too strongly that angina pectoris is a diagnosis based on symptoms and not on physical signs. In about 30 per cent. of cases the diagnosis must be made in spite of a negative physical examination, in the absence of any abnormality of the electrocardiogram, and without any help from rœntgenographic examination of the heart. This fact assumes tremendous importance in relation to individuals who are fraudulently seeking insurance or payments for disability. If a person seeking insurance is dishonest enough to conceal symptoms of angina pectoris, all methods of examination may fail to disclose the existence of coronary sclerosis. If an individual seeking to establish a claim of disability feigns the classic symptoms of angina pectoris, no medical examination may be able to prove the fraudulence of the claim.

The classic characteristics of an attack of acute coronary occlusion are too well known to warrant repetition here. However, one must be on the alert for seizures of pain of short duration and which produce little or no disability, or for attacks in which pain is absent and its place taken by severe seizures of suffocation with or without marked prostration. These attacks often are the signals of acute coronary occlusion, and their significance may not be recognized by the patient or by his physician. The patient's recovery may be so complete that, later, he considers himself in normal health. It is here that the electrocardiogram assumes a conspicuous rôle in the diagnosis, as the relic of the occlusion may persist in the tracing for months, even years, after the event. It is obvious that it is necessary for the examiner to be familiar with the evolution of the electrocardiographic changes that follow acute coronary occlusion, particularly with the late residual changes.

The greatest confusion exists regarding the significance of the electrocardiographic findings in the diagnosis of angina pectoris and of coronary sclerosis. The impression is too prevalent that a normal electrocardiogram rules out the existence of coronary sclerosis and that there are a variety

of changes in the electrocardiogram which positively denote its existence. Fully a third of the patients with angina pectoris have electrocardiograms that are normal in all respects. Except for the typical electrocardiographic patterns that follow acute coronary occlusion, there are no other electrocardiographic changes that positively denote coronary disease. Delays in conduction, either in the bundle of His or in the bundle-branches, or a prolonged Q wave in lead III, as defined by Pardee, are presumptive but not positive evidence of coronary occlusion. Inversion of the T waves, except those that represent a relic of acute coronary occlusion, is not an expression of chronic insufficiency of the coronary circulation but is the result of associated, cardiopathologic changes, the commonest of which is hypertensive heart disease.

In discussing the prognosis of coronary disease I am reminded of Hutchinson's remark that, in medicine, there abideth faith, hope, and charity: "We have Diagnosis, which is a matter of faith; Prognosis, which is a question of hope; and Treatment, which is too often an affair of charity." Mackenzie followed 213 cases of angina pectoris and found that the average duration of life from the onset of symptoms was five and four-tenths years. In White and Bland's series of 486 patients, 273 of whom are living and 213 of whom are dead, the average duration of life following the onset of the syndrome of angina pectoris was four and nine-tenths years. They expressed the belief that, by the time all were followed until the time of death, the average duration of life would exceed Mackenzie's figure. Eppinger and Levine found the average duration of life in their series after the onset of symptoms to be four and five-tenths years.

White and Bland studied the life expectancy of patients in relation to the age of onset of angina pectoris. Starting at forty years of age and grouping the patients by five year periods up to the age of seventy-five years, they found that patients forty years of age at the onset of angina pectoris lived 28 per cent. of their normal expectancy (based on life

expectancy tables of the Metropolitan Life Insurance Company), those forty-five years of age lived 26 per cent., fifty years 21 per cent., fifty-five years, 32 per cent., sixty years 37 per cent., sixty-five years 35 per cent., seventy years 35 per cent. and, those seventy-five years, 21 per cent.

White and Bland[10] also found that the presence of hypertension, gallop rhythm, and poor heart tones had an unfavorable influence on life expectancy. The life expectancy of patients with angina pectoris complicated by coronary thrombosis was almost exactly the same as that for the 213 patients with angina pectoris who were followed until death occurred. These observers found the electrocardiogram of little help in predicting the outcome of the disease. They point out that with proper care, in cases of angina pectoris in which findings are normal on cardiac examination, the outlook for the patient is particularly good.

Eppinger and Levine found that men and women survive an equal length of time after the onset of angina pectoris. When the disease occurred among patients with hypertension, they found its onset to be four years earlier than when it occurred among patients without hypertension. Under these circumstances the duration of the disease was longer, but the age of the patients at death was about three years less than average. They called attention to the fact that patients whose parents lived to an average age of less than sixty years died five and six-tenths years earlier than those whose parents lived past the age of seventy years. They considered that the defect of vascular vulnerability was transmitted more prominently by the female than by the male parent. Obesity had no effect on the age of onset or on the duration of the disease. These observers found that patients whose electrocardiograms were normal had symptoms one year longer but died at an earlier age than the average. The occurrence of negative T waves in lead I or in leads I and II reduced the duration of life one year below the average. In cases in which the electrocardiogram showed defects of con-

duction or distinctly low amplitude, the prognosis was slightly better than the average for the entire series.

Eppinger and Levine also found that the average duration of life after recovery from an attack of acute coronary thrombosis was three and five-tenths years. In White and Bland's series[10], in which only half of the patients are dead, the average duration of life to date is two and four-tenths years. Conner and Holt have expressed the prognosis in their series of 287 cases of coronary thrombosis by a different method; at the end of one year those patients who were alive and well comprised 75 per cent., at the end of two years 56 per cent., at the end of three years 42 per cent., at the end of five years 21 per cent., and at the end of ten years 3.4 per cent.

These findings may be summarized somewhat as follows: A patient with angina pectoris may expect to live four and a half to five years after the onset of symptoms. The fact that an attack of coronary occlusion complicates the syndrome does not materially affect his life expectancy, provided he survives the attack. In general, angina pectoris at any age reduces the life expectancy to a fifth or a third of the normal for the age at which the onset of symptoms occurs. The average duration of life after the onset of coronary occlusion probably is about one or one and a half years less than that observed after the onset of angina pectoris.

In conclusion, I should like to caution against a too easy acceptance of the idea that coronary sclerosis is markedly on the increase. At least another decade of careful study will be required before that question can be answered. Insurance companies will be spared many avoidable losses if it is clearly understood that angina pectoris is a diagnosis based to a much greater extent on symptoms than on physical findings, and that often the diagnosis can and must be made when the latter are entirely normal. Knowledge that the electrocardiogram is often entirely normal in the presence of severe coronary sclerosis should dispel the almost naive regard some examiners seem to have for the electrocardiogram as a

kind of final arbiter in the question of the presence or absence of the disease. Insurance examiners must be alert to the minor manifestations of acute coronary occlusion and recognize that the patient's recovery may be so complete that he considers himself a normal applicant for insurance. Familiarity with the late residual changes in the electrocardiogram following acute coronary occlusion is absolutely essential to the exclusion of this group of high insurance risks. It is axiomatic that careful interrogation of all patients past forty years of age for incidents suggestive of coronary disease constitutes the most effective means of arriving at the diagnosis.

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DR. FRASER—The first discusser of this paper will be Dr. Henry Wireman Cook, Medical Director of the Northwestern National Life Insurance Company. Dr. Cook.

DR. COOK—Dr. Fraser and Members: I am particularly sorry that Dr. Barnes could not be present, as I am sure that we would be interested not only in the presentation of the paper but in his closing informal remarks. (Continued by reading prepared paper.)

While "heart disease" has always occupied a prominent place in insurance medicine and has had frequent discussions in the programs of our medical directors' associations, there has, in recent years, been a marked increase in interest in the subject, and at the same time, at first a gradual and in the past ten years a rapid change in our conception of heart disease, both as a clinical entity and especially in its relation to insurance medicine. Prior to and during the first decade of the century, and even for the second, the problem of heart disease was limited principally from an examiner's viewpoint to the detection of murmurs, and from the home office viewpoint to the evaluation of these murmurs which were roughly grouped for rating purposes into three classes: "functional"; organic mitral, subdivided by history of rheumatism and degree of recognizable hypertrophy; and aortic.

Aside from this consideration of murmurs, heart disease was not an underwriting problem, and in life insurance appeared only in death claim analyses, expressed in the terminology of that time as myocarditis, fatty degeneration, arteriosclerosis, apoplexy, Bright's disease, heart disease, heart failure, and especially acute indigestion, etc. Angina pectoris was a rare disease, well known by the classical descriptions of Heberden and John Hunter, but

entering with extreme rarity into the problem of selection. Coronary sclerosis was relatively unknown, and certainly not considered except as a pathological finding.

It was not until the advent of the sphygmomanometer into general clinical and insurance use that we had our first practical cardiovascular test which could be relied on to detect, in a considerable percentage of cases, changes sufficiently early to permit selection against individuals in apparently robust health, and yet where early claims were being presented from one or more of that heterogeneous group of cardiovascular diseases mentioned above. The change is very remarkable from this attitude of only twenty-five years ago, and the concept which is gradually forming and crystallizing of the major part which coronary sclerosis plays in these tragic and costly death claims in the two decades from 45 to 65, and especially of the belief that we are in possession of a rapidly growing knowledge and mechanical aids which permit a relatively early recognition of the condition. Of the many recent papers on blood pressure, cardiac radiology and measurement, and electrocardiography, both clinical and statistical, I know of none that is more helpful than this one of Dr. Barnes', nor of the clinical papers none that is more understanding and appreciative of our underwriting problems. Not infrequently our clinical papers are too unrelated to underwriting, and I want to especially thank Dr. Barnes for having kept so consistently and so helpfully in mind this, for us, necessary relationship. Dr. Barnes truly says that many of our difficulties are due to a misunderstanding between the clinical and insurance viewpoints, and he has done much to clarify this misunderstanding.

Whether coronary disease is increasing as rapidly among the general population as has seemed apparent, is for us largely of academic interest. From its astonishing frequency in claims as the cause of death, it may in part represent the attending physician's intention to establish his reputation as informed and up-to-date. Coronary disease would be unique as a new conception if it were not over-emphasized. Nevertheless, waiving aside differences of opinion on this subject, there can certainly be no question that cardiovascular disease is today in the life insurance

business the overwhelming major factor in profitable selection, and especially in applications for larger policies,—$10,000 and over.

Coronary sclerosis and cardiovascular diseases compel our interest from the fact that they cause over 400,000 deaths in this country annually, that they are the largest factor in both general and insured mortality, that there are several million established cases of insurable age in varying stages of the disease, and that of 100 large claims totalling over $70,000,000 recently analyzed and reported to this Association, 28 died of cardiovascular disease within five years of their examination by a number of class A examiners, and accepted as standard risks.

As my interest in cardiovascular disease is so largely in relation to insurance experience and selection, I will be pardoned for bringing this discussion back to that problem and for outlining, what seem to me, the factors which the practical underwriting of risks requires in relation to this particular impairment.

1. To accumulate and become familiar with current literature on the subject and modern methods of diagnosis, and as fast as practical to supplement this with statistical experience as soon as accurate data in sufficient volume can be accumulated and analyzed. This has already been done for blood pressure and will be available for X-ray plates of heart and for electrocardiograms.

2. To convey to our examiners as rapidly as possible correct methods of cardiovascular examination and correct interpretation of findings, both subjective and objective. The weakest link in our chain is the average examiner. He does not appreciate the significance of suggestive symptoms and signs of cardiovascular disease. He often does not uncover them and often does not report them when he does find them. I have heard medical directors say that they do not care for the examiner's opinion. "Give us the findings and we will do the interpreting." I think they overlook the fact that these signs and symptoms are not so obvious that all who run may read. They can only be read by one who has an *understanding* vision. Unless cardiovascular disease is understood by the examiner, its signs will be overlooked

or discounted. I have seen good men overlook a thickened tortuous artery, a split first sound, a soft murmur, a displaced apex, shade a blood pressure 20 points or more or give the lowest reading obtainable. A corps of alert, informed examiners, who are so loyal to the home office that they will give every sign and symptom obtainable, is, I believe, the fundamental requirement of sound selection, and it is of great value to obtain from them an informed and intelligent opinion of cardiovascular signs and symptoms. Mediocre examiners who feel subject to the caprice or favoritism of agents are one of the chief factors in an unfavorable experience, especially in large risks and in cardiovascular disease. It takes a well-trained man to find the evidence, and a courageous one to report the borderline signs and symptoms, and it is these we need. We cannot expect him to be popular with the questionable or uninformed agent.

3. To give in home office decisions greater weight to the significance of lesser degrees of hypertension, both systolic and diastolic. Beyond question beginning hypertension often gives us a warning signal years in advance of any other sign or symptom obtainable in an insurance examination. We must keep in mind the opinion of Alverez and Stanley after a study of six thousand blood pressures from 15 to 84 years of age, that "a pressure of 115 mm. Hg. is just as normal, and a pressure of 140 mm. Hg. is just as abnormal in an old man as in a young one". Dr. Hunter ten years ago pointed out that "average" blood pressures at various ages do not represent "normal" blood pressures.

4. To interpret more strictly and more in line with insurance experience and less in accord with clinical opinion the data past and present which suggest cardiovascular disease. Some of our costliest mistakes have been on large risks with insurance records of heart murmurs which eminent cardiologists regard as "functional", on hypertensions which are "cured" by removal of a focus of infection, by a rest cure, by alimentary purification, etc., and on cardiovascular suspects where some pet functional test of our own or some new diagnostic instrument, such as the

X-ray or electrocardiogram, is negative and is interpreted as having excluded organic disease. It is equally satisfying and exhilarating to both cardiac patient and to physician to offer authoritative optimistic prognoses. The survivors extol the virtues of the physician, but the silent majority provide a costly underwriting experience. We had a recent illustrative case. The applicant's last policy was taken in 1927, but since then he had had an annual health examination. All were entirely negative until May of this year, when he complained to the examiner that he had substernal discomfort on exertion. The examiner happened to be an internist, and as the man asked for a full clinical examination everything was done,—functional tests, X-ray, electrocardiogram, etc. All were entirely negative. Our examiner wrote us a long letter, ending with his opinion that the condition was "functional"—an effort syndrome, as he diagnosed it. We paid the claim in August from angina pectoris.

5. To view cardiovascular disease in middle age or beyond as the generalized aging process which it is, rather than in its insular textbook classifications as a localized condition subject to curative or corrective treatment. Chronic degenerative disease of the arteries of the brain, of the heart, of the kidneys, of the pancreas, are not sufficiently regarded as localized manifestations of a widespread process. It is too much to expect that men in the sixth decade who have had clinical or insurance records of heart murmurs, irregular hearts, hypertension, albuminuria, cylindruria, glycosuria, should become actually, as they do on paper, standard risks, when they suddenly are persuaded that they need $1,000,000 of insurance, and I believe that holds true, no matter whether they were examined by the best known of internists or had entirely negative plates and tracings from the best laboratories. Dr. Barnes comments on our naive acceptance of negative findings. I have three intimate friends who have survived their first coronary occlusion, all of whom could today pass the most searching medical examination. If this is true for a major manifestation, I believe it is also true for the less dramatic signs and symptoms of cardiovascular disease. Examiners must be taught to recognize suggestive signs

and symptoms and we must learn to evaluate them. Early and even advanced cardiovascular disease of this degenerative type is extremely difficult to detect. Often these men may appear in robust health and have only slight subjective symptoms, i.e., substernal discomfort or pain or slight dyspnea. This robust type of individual often, however, has some elevation of blood pressure,—systolic over 138 and diastolic over 92.

There is another group that I believe deserves our careful consideration,—the man of 50, 55, 60, who does not look quite up to par, although the examiner may find no gross lesion. He may look nervous and apprehensive, his muscles may be flabby; he may have lost weight; he may have shadows under the eyes; his arteries may be moderately thickened or tortuous and visibly pulsating, although his blood pressure may be at or below 136. However, there may be an old record of hypertension. His retinal vessels might show changes. His heart sounds may not be quite clear, the first sound without a definite murmur, but impure, perhaps a soft so-called "functional" murmur, or an occasional extra systole. (For underwriting purposes the "functional" heart murmur over 40 is non-existent.) His complexion may be a little sallow, perhaps an arcus senilis. Nothing very definite perhaps. The average examiner answers all questions in the negative. Questioning may reveal that he tires more easily, plays 9 holes of golf instead of 18, is nervous, smokes to excess, has bad business worries, or personal troubles, sleeps restlessly, perhaps has some indigestion. This group will have an unfavorable experience. Such a man has not a normal chance to survive a pneumonia or even an automobile accident, nor to normally sustain the stress and strain of modern social, industrial, or professional life.

In conclusion, I believe we are relatively at the beginning of a truer conception of chronic cardiovascular disease, but that with a better knowledge of its pathology, with the aid of modern instruments of precision, and with a carefully analyzed insurance experience, our clinical understanding will rapidly clarify, and our underwriting at ages 45 to 65, especially for larger amounts, will be considerably more profitable than it is at present.

DR FRASER—The next discusser of this important paper is Dr. William Muhlberg, Medical Director of the Union Central Life Insurance Company. Dr. Muhlberg.

DR. MUHLBERG—Mr. President and Gentlemen of the Association: (Read paper.)

There is perhaps no subject in the field of medical selection that has been quite so perplexing as that presented by the problem of angina pectoris and coronary artery disease. The rapidly growing incidence of reported deaths, especially of coronary artery disease, is particularly challenging. Having no statistics to fall back on, because most of the progress in the knowledge of this affection has been made within recent years, we must necessarily rely almost entirely on the advice and recommendations of our cardiologists.

The Association is very fortunate in having a nationally known cardiologist, Dr. A. R. Barnes, present his views. His statistics giving the expectancy of life of persons having had angina pectoris or coronary occlusion are most valuable to us, particularly for purposes of calculating the reserves to be set aside for disability claims. Most of us, too, will agree with Dr. Barnes that statistical studies indicating a marked increase in the incidence of coronary sclerosis and heart disease must be carefully checked by post-mortem findings, the age incidence of the population, and the tendency of doctors to include deaths in the heart group that were formerly charged to apoplexy, Bright's Disease, and senility.

Unfortunately for me, Dr. Barnes has so adequately and authoritatively dealt with his subject that he has left me very little opportunity for discussion. Nevertheless it seemed to me that I might contribute something of interest to the Association by procuring a cross-section of opinion of cardiologists throughout the country, particularly those who have consented to undertake our electrocardiographic examinations. Since our decisions are largely based on the reports of these specialists, it might be well to have some idea of their views. Accordingly, I sent questionnaires to these specialists and received 102 responses.

The use of questionnaires is not always a satisfactory way of getting first-hand knowledge. Personally, I dislike them; I was, therefore, much gratified with the courtesy displayed and the care with which the questions were answered.

In response to my first question, which read:—"Do you believe that the family history is significant in predisposing to coronary artery disease?"—the opinion was almost unanimous that the family history is of great importance, although some felt that it was more significant for angina pectoris than for coronary artery disease.

The next question:—"If you believe the family history is significant, what diseases in parents, brothers or sisters are of importance?" There was necessarily some difference of opinion with reference to the order in which certain diseases were mentioned, but the following list indicates in order of importance, the diseases referred to: Arterio-sclerosis, degenerative heart disease, hypertension, chronic nephritis, apoplexy, angina pectoris, diabetes, sudden death, so-called acute indigestion, overweight, premature senility, and syphilis.

The next question read:—"Does there appear to be any causal relationship between coronary artery disease and previous illnesses or co-existing illnesses, such as glycosuria, gall-bladder disease, renal colic, focal infections, cardiac valve lesions, etc?" There were 14 negative replies, and 9 answered "Yes". 50, or about one-half, felt that glycosuria or diabetes played an important part, and 21 mentioned gall-bladder disease. Somewhat to my surprise, only 15 thought that focal infection was significant, and 6 of them felt convinced that it was of no significance. Only 6 mentioned syphilis, 3 rheumatism, and 4 believed that general infections, such as previous typhoid fever, etc., were of importance.

The next question read:—"Insurance statistics indicate that high blood pressure, overweight, rheumatism, albuminuria, glycosuria, cylindruria, syphilis, alcoholism, irregular or intermittent pulse, or strenuous modes of living predispose to higher death rates from heart disease. Do you believe they also predispose to coronary artery disease?" 46 answered—"Yes". 4 either

answered—"No" or left the question unanswered; and 42 others stated they felt that some of these impairments were of significance and others were not. Their replies in order of choice were as follows:—

Blood pressure	41
Glycosuria	36
Overweight	32
Strenuous modes of living	30
Syphilis	20
Albuminuria	14

In answer to the question, "Do you believe that the immoderate use of tobacco, tea, coffee, or such drugs as aspirin, have a predisposing influence?"—57 answered "No". 4 stated, "I do not know". 17 thought tobacco had some influence. 21 were rather doubtful, and 3 felt that it aggravated conditions. Only 5 believed that tea and coffee played any part. 8 were doubtful. None of them believed that drugs were of importance.

With reference to metabolic conditions, it was asked:—"Some pathologists believe that disturbance of internal secretions, especially of the thyroid, adrenals or cholesterin metabolism play a part. What is your opinion?" The answers to this question are rather interesting, particularly in the light of the important research work that is at present being conducted particularly in cholesterin metabolism. 7 of the respondents stated "Yes". 10 preferred to express no opinion. 14 felt that such relationship was as yet unproven. 6 were doubtful. 4 stressed the importance of hyperthyroidism; 3 of hypothyroidism; and 7 dysthyroidism. 24 were convinced that some disturbance of cholesterin metabolism played an important part.

The next question was:—"There have been suggestions in medical literature that an absence of some of the vitamins, particularly Vitamin G, may predispose to arteriosclerotic conditions. Do you believe that this might play a part?" Almost unanimously the response was that vitamins either played no part or that not sufficient evidence has as yet been brought forward to make such an etiological factor a probable one.

The next question read:—"Medical Directors are inclined to the view that angina pectoris and perhaps coronary disease are more often encountered in persons in the higher economic stratum, especially those who are subjected to the burden of heavy responsibilities with attendant worries, anxieties, nerve strains, etc. Do you believe that these factors play a part in predisposition?" 77 answered this question "Yes". 9 answered "No", and 14 stated that they were in doubt or not convinced. A few expressed the opinion that the economic status played less part than worries, anxieties and emotional stress, that are perhaps more often encountered in the upper stratum. Quite a few believed that angina was more likely to be influenced in this way than coronary disease.

The next question read:—"In a general way, what has been the percentage of your cases of coronary disease, in whom high blood pressure existed? Or thyroid disease? Or a history of strenuous athletic or physical exercise?" In answering the first question, namely high blood pressure, the estimates ran from 25% to 90%, with an average of about 60%. However, the majority stressed the fact that not a few cases developed coronary disease where previously the blood pressure had been normal or even below normal, systolic and diastolic. Many were of the opinion that blood pressure caused arteriosclerosis with secondary involvement of the coronary artery. With reference to thyroid disease, the great preponderance of opinion was that there was very little relationship between thyroid disturbance and coronary disease. 5 mentioned cases where they felt the condition was due to hypothyroidism or myxedema, and 2 had seen cases due to hyperthyroidism. These replies are very significant, in view of the well-established relationship of hyperthyroidism and other forms of heart disease and the frequent association of myxedema and hyper-cholesteremia with premature hardening of the arteries. With reference to a history of strenuous athletic or physical exercise, 49 answered "No". 8 felt that there was some significance; the majority expressed the opinion that exercise often precipitated acute attacks of stenocardia, where the coronary disease already existed. Quite a few were convinced that too much exercise past the age of 40

or 50 was dangerous and should not be indulged in by men unaccustomed to it previously. Those who felt that exercise or hard work played a prominent part qualified their statements by indicating that most of their patients were seen in clinics or hospitals among laborers and that the relationship might have been solely incident to the occupation of such groups.

Previous Symptomatology—"What in your experience have been the dominant symptoms that might point to coronary artery attacks later in life?" 21 felt that there were no premonitory symptoms; 80, however, chose various symptoms and signs, of which the most important in the order of frequency mentioned are as follows:

31—occasional tightness in the chest, or substernal pain.
26—shortness of breath or dyspnoea after mild exercise that formerly caused no respiratory distress.
25—indigestion or bloating after meals.
18—hypertension.
13—slight pain of the anginal type following effort.
13—fatigability.
12—excitability, nervousness, worry and anxiety.
8—overweight.

There were several who mentioned the fact that there are certain types who seem to be predisposed to coronary artery disease—namely, the overweight, who over-eats, has occasional sugar in his urine, with a tendency to hypertension, subject to outbursts of anger, who suffers from bloating after meals and is easily fatigued.

Electrocardiograms—"What deviations from the normal in the electrocardiograms would you consider most significant in predicting the probability of future coronary artery disease?" This question was badly worded, the intent being to develop information with reference to persons who probably had coronary disease, but who as yet had not shown any symptoms. Fortunately, practically all of the cardiologists understood the intent of the question, and in answering, qualified it as indicated. There were 26 who thought that if coronary disease had as yet no

caused any pronounced symptoms or had not led to myocarditis or thrombosis or infarction, the electrocardiogram would probably be practically normal. Others indicated some abnormality in the electrocardiogram, depending upon the extent of the sclerosis of the coronary artery or the progress which the disease had made. The various abnormalities indicated, often of course, in combinations, were as follows: Practically no changes in the P-wave or the P R interval; a widening of the QRS was mentioned 12 times; low voltage 23 times; notching or slurring of the QRS complex 25 times; a deep Q3, 26 times; changes in level of the R T interval, 14 times; high takeoff, 4 times; low takeoff, 2 times; evidence of the Pardee coronary cove, 8 times; inversion of the T wave, particularly T I and T II, were mentioned 36 times; and a few thought that even inverted T III was significant, particularly if T I or T II were of rather low voltage. A low voltage of either T I or II was mentioned 17 times; left axis deviation, 13 times. Many of the cardiologists stressed the importance of the 4th lead and believe that this, with further study, would probably be of more value than any of the other leads. It is evident that many of the cardiologists believe that the electrocardiogram is of real value, even before there are manifest symptoms. It is, however, of importance to note that even those who indicated what they considered of significance in the electrocardiogram issued warnings that the electrocardiogram may be perfectly normal, even in the presence of a well-advanced coronary artery disease.

(b) "What deviations in the electrocardiagrams are most likely to be found in persons who have recovered from attacks?" The answers to this question were of a great deal of interest, because there appears to be practically a unanimity of opinion among specialists as to the findings to be expected in these cases. Again there was the admonition that in some instances, in as short a time as six months after a fairly sharp attack of occlusion, the electrocardiogram might be entirely normal. As a rule, however, there were persistent evidences for many years. 102 questionnaires were returned and this question was not answered in two instances. The abnormalities mentioned, and of course, often

in combination, were as follows: Prolongation of the P R interval in 5 times; low and wide QRS—17; low QRS—12; wide QRS—12; notching or slurring of the QRS complex—32; bundle branch block—12; deep Q III—30; deviation from the isoelectric of the R T interval—26; high take-off—2; low take-off—3; Pardee's curve—18. Changes in T were mentioned 79 times and in practically every instance, inversion of the T I or T II, or both were to be looked for; and again the importance of Lead IV was insisted upon by many of the specialists.

X-Ray—(a) "Do you consider the x-ray of value in diagnosing coronary artery disease? If so, what evidence would you regard as important?" 47 answered "No". 4 answered "Yes". 40 stated that it was of value, not in diagnosing coronary artery disease directly, but in getting valuable information with reference to hardening of the arch of the aorta, aneurism, or cardiac enlargement, etc. 7 felt that it was of some value in detecting calcification of the coronary artery, but that there was much needed in the way of more perfect technique before this becomes a very valuable method of diagnosis.

(b) "Do you believe that the orthodiagraphic or fluoroscopic method is preferable to the photographic?" 49 believed the orthodiagraphic was more valuable. 14 were divided in their opinion, and the others were in favor of the photographic method. But many who favored the photographic method stated they felt the orthodiagraphic or the fluoroscopic method might be of greater value, if done by one skilled in the use of these methods.

(c) "If limited to a single photographic method, do you prefer the antero-posterior or the oblique exposures?" The opinion was almost unanimous that the anteroposterior was preferable, although many indicated that they used both. 8 of the specialists preferred the oblique method, principally because it gave a better shadow of the aorta.

While the questionnaire developed no information that is essentially new to members of the Association, nevertheless it is somewhat reassuring to know that the opinions of cardiologists throughout the country are practically in harmony with our views.

DR. FRASER—The next discusser of this paper will be Dr. Benjamin M. Baker, Jr. of Baltimore, who so kindly offered to read the paper in the absence of Dr. Barnes. Dr. Baker.

DR. BAKER—Mr. President and Members of the Association: (Read paper.)

We have had the good fortune to hear an intricate medical topic discussed in a clear, concise and forceful way. The diagnosis of disease of the coronary arteries is one of the most important and difficult problems the physician is daily called upon to solve in his practice and to insurance examiners and officials it presents in addition many perplexing and amazing questions. I am impressed by Dr. Barnes' intimate knowledge of the subject, but what has appealed to me especially is the gracious spirit of humility with which he has presented it. He has put the matter frankly before us and clearly has pointed out the difficulties and uncertainties which surround it.

Dr. Barnes has remarked that the medical profession is criticized for being unfair to insurance officials on the subject of coronary sclerosis. The truth of the statement cannot be challenged but in justice its implication should be softened. Insurance companies must not expect the impossible. In many instances medical examiners' mistakes are inevitable. Only when the gaps which exist in our diagnostic resources are bridged will the recognition of coronary sclerosis by medical examiners reach a satisfactory average.

Dr. Barnes has impressed upon us the fact that there is no reliable information to substantiate the common contention that coronary sclerosis is on the increase. Thirty years ago men in robust health unexpectedly dropped dead of coronary occlusion, just as they do today, and no doubt these tragedies will continue to occur. Often there are no preceding symptoms of heart disease; at other times physicians fail to recognize the grave significance of what appear to be but trivial discomforts. However, it is only fair to assume, in defense of physicians, that now they are more "coronary sclerosis wise" than they were

thirty years ago, and therefore Angina Pectoris is on the increase because it is recognized more frequently.

In any discussion of Angina Pectoris it is essential in order to avoid confusion that there be a clear understanding of the meaning of the term. This necessity arises from the fact that physicians use the term in a different and often even contrary sense. One group regards it merely as a symptom complex due to a multiplicity of causes and without a uniform anatomical basis. Another insists that it is a definite disease and that the symptoms are always due to a disturbance in the blood supply to the heart. Still another group, comprising the majority of physicians is somewhat uncertain about the exact meaning of the term. For the purpose of this discussion I shall define Angina Pectoris as the clinical manifestations of an inadequate coronary circulation.

We have heard the diagnosis of Angina Pectoris discussed so fully that it seems almost superfluous to say more about it; nevertheless, the accurate diagnosis of Angina Pectoris is so important that repetition for the sake of emphasis cannot be out of place. It may profitably be emphasized again and again that the only manifestations of Angina Pectoris which justify the diagnosis consist of the patient's symptoms. Without subjective sensations the diagnosis is impossible and more important still these sensations and these alone may be present. There are no objective signs which are distinctive of Angina Pectoris; indeed, objective signs and positive information supplied by instruments of precision may be entirely lacking. When signs are present they merely corroborate the diagnosis but alone they do not establish it. Therefore, when the diagnosis is made it must result from the physician's appraisal of the account the patient gives of his symptoms. This creates an extraordinarily difficult situation. The physician must not only carefully analyze the story but he must shrewdly judge the patient. He must determine what the patient means by the sensations he describes. One flinches at the merest approach of pain while another with singular fortitude makes light of it. What is described as agony by one is belittled by another. A person in close associ-

ation with a sufferer from Angina Pectoris has had the symptoms of the disease, and their significance deeply engraved upon his mind. For him the tragedy of sudden death may tinge every trivial sensation with fear and the anticipation of life's dissolution. The physician then must be a keen observer of his patient's most intimate human reactions and it is here that experience has its rewards. The young physician recognizes readily a valvular heart lesion but in the diagnosis of Angina Pectoris the advantage goes with the wisdom of age. Too often the young physician feels that he can rely upon the electrocardiogram or the roentgen ray to compensate for his lack of experience. But the fact is too plain that galvanometric clinicians are of no use to medical directors of insurance companies.

The general run of medical examiners cannot be made up of physicians either properly trained or willing to give the necessary valuable time to make as many correct diagnoses as might be made under ideal conditions. Another obstacle is the lack of integrity of some patients who apply for life insurance or demand compensation. An otherwise healthy man need not tell the examiner that on the hilly eighteenth hole of his favorite golf course, he has to pause for a brief rest because of a disagreeable tightness beneath the sternum. If a dishonest policyholder wishes to claim disability upon the basis of Angina Pectoris, easily available professional coaching can perfect malingering to a point where it will elude the most searching of modern scientific medical examinations. All of our modern diagnostic tricks fail to resolve the difficulties presented by the frequent occurrence of these deceits.

At the suggestion of your President certain observations which have been made during the past several years upon a possibly precise diagnostic test for Angina Pectoris may prove of interest to you. All theories which relate the pain of Angina Pectoris to coronary insufficiency accept the assumption that the provocative factor is myocardial ischaemia. In the monograph of Keefer and Resnik the hypothesis is presented and supported that the fundamental factor is depriving the myocar-

dium of a normal oxygen supply. It is at once apparent then that this condition can be easily reproduced experimentally.

In August, 1933, Rothschild and Kissin published exceedingly interesting observations upon the clinical application of this principle. In two groups of individuals they induced generalized anoxaemia by re-breathing. One group of twenty-six patients was considered to have an impaired coronary circulation and another of twenty patients was thought to have a normal coronary circulation. Of the control group of patients without an impaired coronary circulation none developed pain as a result of re-breathing; of the twenty-six patients clinically diagnosed as having coronary sclerosis, eighteen developed attacks of pain indistinguishable from their spontaneous attacks. More recently Katz, Hamburger and Schutz have studied the effect of general anoxaemia induced by re-breathing upon the S-T segments and T waves of the electrocardiogram in normal persons and in a number suffering from Angina Pectoris. In practically every instance of seventeen normal subjects tested there was depression of the S-T segment or flattening of inversion of the T wave but without the development of pain. Of six subjects with Angina Pectoris who were tested, four developed these electrocardiographic phenomena of spontaneous attacks of Angina Pectoris without the pain and these authors concluded that induced anoxaemia is not a reliable and perhaps even a dangerous test for Angina Pectoris.

Our experience with the effects of induced anoxaemia began in 1931 when an elderly patient with generalized arteriosclerosis and moderate hypertension related that he had experienced repeated attacks of typical Angina Pectoris for the first time in his life while on a recent visit to a high altitude. As soon as he returned to sea level he pursued the activities of an average business life without discomforts. Induced general anoxaemia caused a reappearance of precisely the same symptoms he had experienced at the high altitude.

During the last three years we have produced anoxaemia in a group of patients who have complained of pain in the region of the heart. The majority of these have been seen in private

practice and a clear cut intelligent history served as the basis for deciding whether or not they had true Angina Pectoris. Two independent opinions have always been obtained before the test has been made and the second opinion has always been that of a distinguished and experienced clinician. All patients who have obviously had a coronary occlusion have been excluded. A number of the patients considered as having true Angina Pectoris have had no objective manifestations of cardiac disease and their stories alone have served as the basis for the accepted diagnosis. The group of patients complaining of pain over the heart but considered not to have Angina Pectoris has included such conditions as effort syndrome, spinal arthritis, rheumatic heart disease, malingering and anxiety neurosis. No case has been included in either group when the diagnosis was uncertain.

The test has been carried out in an exceedingly simple way. The patient rebreathed from an ordinary basal metabolism apparatus, the tank of which had been previously filled with two or three liters of room air. In some tests the soda lime was left in the circuit; in others it was removed. The patients have all been reassured of the harmlessness of the test and have been told that it would be stopped when their discomfort became too great. The possibility of pain production has, of course, not been suggested, and nervous patients have been allowed to go through a preliminary period of breathing oxygen. When pain has resulted it has always disappeared rapidly as soon as the patient breathed room air or pure oxygen and only once or twice has it been necessary to hasten relief by giving a vasodilator. No patient has become unconscious from oxygen want and there have been no other untoward effects.

The results of these tests may be stated briefly. In none of fourteen instances did pain result when the test was applied to a patient thought not to have Angina Pectoris. In all but one instance, pain which the patients stated was precisely like their spontaneous attack was reproduced in the group composed of fourteen patients assumed to have Angina Pectoris. The failure of the test occurred in the second patient tested. He was a young man of thirty-five who was extremely apprehensive and

refused to continue the test as long as the average time tolerated by other patients in this group. He has since died of coronary occlusion. As previously stated the patients were selected with great care. If then it be accepted that the diagnoses are correct the test does not give pain to patients who do not have Angina Pectoris. Of those who have Angina Pectoris it reproduces pain in 93%.

It is not the object of this discussion to advocate unjustifiable reliance in the test described. The diagnosis of Angina Pectoris still depends upon conclusions reached after a physician has analyzed a patient's story. At present a clinical diagnosis is serving as a basis for evaluating the reliability of the test described. If the experience we have thus far had continues then, and then only, will the test be of real value and several years of carefully following the patients tested alone can settle that point. Nevertheless, even now the test should greatly facilitate the detection of applicants who do not divulge to the medical examiner certain symptoms which would suggest the possibility of Angina Pectoris. In some instances, at least, the test assists in the detection of malingering.

DR. FRASER—Dr. William Bartlett, Medical Director of the John Hancock Life Insurance Company, will continue the discussion.

DR. BARTLETT—Gentlemen, I am going to be very brief, but just before I left the Home Office, the Actuary handed me certain figures that had been compiled in reference to coronary disease other than angina pectoris, and they were so surprising to me that I thought I would read them to you.

These figures are the adjusted death rate per 100,000 lives exposed on Industrial premium paying business. In 1930 there were 8 deaths from this cause; in 1931, there were 10; and in 1932 there were 13; and in 1933 there were 17.3, an increase of 116% since 1930, which was the first year that this cause was separated from the general group of heart disease or angina pectoris.

Now it seemed to us that this astounding increase in the diagnosis of disease of the coronary artery might have been at the expense of the diagnosis of angina pectoris. We therefore took the death rate from this cause, which was as follows:

1930	11.6
1931	11.2
1932	10.9
1933	10.2

showing a decrease of 12%.

Now in 1930, the first year these death rates were compiled, we had 293 deaths from angina, from coronary sclerosis, in the Industrial branch. In 1933 we had 650 deaths from that cause. Now I don't believe for a minute that that represents an increase in coronary sclerosis and am not presenting it for that purpose, but it is interesting to inquire from what group those deaths are coming, and that is what we are going to try to determine. Thank you.

DR. FRASER—The next subject open for a discussion will be the paper to be presented by Dr. William Bolt, Medical Director of the New York Life Insurance Company, on "Hypertension as an Underwriting Problem". Dr. Bolt.

DR. BOLT—Mr. Chairman and Gentlemen: (Read paper.)

HYPERTENSION AS AN UNDERWRITING PROBLEM

By William Bolt, M. D., C. M.
Medical Director, New York Life Insurance Company

The prognosis in Hypertension is very important, especially in insurance medicine, yet it is something about which very little accurate information is available in medical literature. Textbooks of medicine have little to say on the subject and it is only within recent years that medical literature contains any reports of series of cases followed for a period long enough to justify conclusions regarding prognosis.

High blood pressure, of course, has been of great interest to medical directors for some years, and since the publication of the Medical Impairment Study, this interest has been intensified because of the results reported therein. Everyone expected that the higher blood pressures would show a higher mortality, but it came as a shock to find such a high mortality for blood pressures which many up to the present had regarded as normal.

Mean Blood Pressure

Before agreeing with the various solutions suggested for handling this problem, it was decided in the New York Life to study carefully the blood pressure material which was available, with the idea of finding out, if possible, the explanation for the blood pressure mortality as reported in the M.I.S., especially in the lower overaverage groups. To begin with, an actuarial study was made of the mean arithmetic blood pressure (one-half of the sum of systolic and diastolic) of standard and substandard material for issues of 1923 and 1925 to 1931, inclusive. The results of this study are shown in Table I and Table II. It is important to remember that this study is based on the arithmetic mean of systolic and diastolic blood pressures, each obtained from the *average* blood pressure recorded on the actuarial cards and to realize that

this average blood pressure is the average in many cases of two or more blood pressure readings taken at the time of the medical examination when the policy was issued. While the use of the mean blood pressure is a new departure in mortality studies, yet the fact that this mean is obtained from the average systolic and diastolic recorded led us to believe that the results obtained from such a study would in general parallel the information obtained from the M.I.S.

Table I deals with the standard issues and Table II with substandard issues. The standard section is, of course, much larger than the substandard and, in general, shows a favorable mortality with the exception of the high group, i.e., 16-24 mm. above the mean. It is interesting to note that the relative mortality in the overaverage (+5 to +15 mm.) group was 116% for standard cases, whereas in the substandard group it was 174%. The relative mortality in the high group of standard cases was 151% and in the substandard class 239%. Although the only impairment recorded on the actuarial cards in both these groups was the blood pressure, there must obviously have been some other factors in addition to the blood pressure which influenced the selectors when deciding the ratings. The experience in the substandard class shows a fairly uniform increase of seventy points in mortality for each ten-point increase in the mean blood pressure, whereas the results given in the M.I.S. show rather irregular increases. While the mortality was naturally higher in this group of New York Life substandard issues than in the M.I.S. (since five points in the diastolic are more important than five in the systolic), the results tend to confirm the M.I.S. findings that there was a decided increase in mortality in all these groups, even where the blood pressure was only 5-15 mm. above the average blood pressure. It should be remembered, however, that both results were obtained by averaging all the blood pressures found on examination.

Factors Affecting Mortality

We felt that the method of judging a blood pressure case by the average of the blood pressures on examination, with no reference to past impairments, might be responsible in part for the

mortality obtained both in the M.I.S. and in the above data. Whether this was so or not, we were of the impression that there must be something in these cases to explain the mortality other than the actual blood pressures found at the time of the examination. It was decided, therefore, to study in detail the actual application papers of those individuals recorded as blood pressure cases from 1923 to 1928, inclusive. Records of all the standard material of 1923, part of the standard material of 1928 and all the substandard material of 1923 to 1928, inclusive, were available. We did not go beyond 1923 because the further we went the less certain we were of the accuracy of the blood pressures recorded. By beginning in 1923 and carrying this study to the end of 1933, there was a maximum exposure of ten years, and by stopping at 1928, a minimum exposure of five years. There is, of course, the objection to results obtained in this way that they do not represent the ultimate mortality, but reference to the M.I.S. will show that the relative mortality of blood pressure cases for the period eleven to nineteen years from issue (213%) was no worse than for the period six to ten years (222%). The mortality ratio for years one to ten (186%) was practically identical with that for all the years one to nineteen (187%). It was felt, therefore, that if we could get a clear idea of what happened in the first ten years we could assume that the experience after that period would not be radically different.

In 1932 Mr. E. W. Marshall drew attention to a number of factors which may influence mortality studies. Any of the points mentioned by him might have affected the blood pressure studies, but two seem worthy of particular attention. These are, first, the effect of changes in standards of selection, and, second, the question of whether or not the material is homogeneous. Most people will agree that in the last ten years our standards of selection have improved. Closer supervision of examiners, more practising physicians familiar with the blood pressure technique from their medical school days and a better knowledge of the importance of high blood pressure and its relation to other impairments on the part of medical directors may in time improve the mortality experience. As an indication of our changing ideas about blood

pressure, it may be mentioned that in revising the material which is the basis of this paper it was obvious that an appreciable number of cases accepted standard ten years ago would now be issued substandard and many granted substandard insurance would now be declined. That this was not peculiar to this Company alone is shown by the fact that the medical directors of two other large New York companies, while investigating their own blood pressure material, were impressed with this same fact.

Blood Pressure Variability

It was decided to make a detailed study of our blood pressure material, with special reference to its homogeneity, and to consider *all* blood pressures recorded instead of only those on the medical examination at the time the policy was issued. A review of the literature focused our attention on some points not wholly new which may have some bearing on the problem. To begin with, there is the variability of blood pressure, both systolic and diastolic. This fact is well known, yet it does not seem to have received due consideration in the estimation of blood pressure in insurance medicine. Text-books state that the systolic pressure of a man in his twenties may vary twenty millimeters in a day. Luckily, with the average man the variation is not great enough to affect his insurability. With the hyptertensive individual, however, the situation is different. Lang states that fluctuations in the systolic may be 50 to 60 mm. and may even reach 100 in hypertension cases. The diastolic varies up to fifty millimeters, the average being twenty-one. Ayman states that in general the proportionate variation of the diastolic is as great as that of the systolic, e.g., in one person with a blood pressure ranging between 160 and 240 systolic, the diastolic ranged from 100 to 150, a variation of one-third in both systolic and diastolic. Hines and Brown, of the Mayo Clinic, in a report on the effect of cold in determining blood pressure variability, state that in an average person recumbent for fifteen minutes the blood pressure falls to the basal level, though it may take forty-five minutes for it to fall to the basal level in a hypertension case. The point is, however, that

ultimately the blood pressure does go down to the basal level even in hypertension cases.

In this connection we were impressed with one factor once we had started our study of the original application papers, and this factor became more evident as the study proceeded. Theoretically, there may be some ground for assuming that the best way to gain an idea of a person's blood pressure is to obtain a large number of blood pressure readings. Practically, however, from the standpoint of insurance underwriting, this idea is misleading. In the great majority of cases blood pressure reviews are favorable to the applicant and consequently tend to give a false picture to the company which places as much weight on these lower readings as on the original high blood pressure. Where the initial blood pressure was high, subsequent blood pressure readings were almost invariably lower. This we found to be true all through our study of both standard and substandard cases. The only exception was when an individual had a record of high blood pressure in the past. In such cases the initial blood pressure on our examination was likely to be lower than the one previously reported, and it was only when further readings were made that the blood pressure was found to be more in keeping with the past history. Our experience indicates that once an individual has been found to have high blood pressure, his next examination for insurance is likely to be arranged under circumstances favorable to him.

What has been said about the variability of blood pressure will explain why this is possible. A person whose cardiovascular-renal condition has not already progressed to the stage where the blood pressure is fixed at a high level can usually arrange to be examined when the blood pressure is at a lower level. Examinations made early in the morning before the daily activities have caused the blood pressure to rise, or made at some other time preceded by a period of rest, are very likely to produce blood pressures favorable to the applicant.

It was felt, therefore, that more attention should be paid to the highest blood pressures reported rather than the average of all the blood pressures found at the examination. Many of these so-called blood pressure cases had had prior blood pressures

which were much higher and had had other conditions which, because they were not present at the examination, had been disregarded. Nowadays we consider a past history of any major impairment important in studying a case, so it did not appear reasonable to us to ignore previous higher blood pressures, previous renal impairments (such as albumin or casts), previous findings of sugar, previous cardiac conditions (such as rapid or irregular pulse, heart murmurs, etc.) and overweight in the past or at the examination. As the purpose of this study was to find out something about blood pressure, we decided to put in a separate group all cases which had had at any time any other impairment beside high blood pressure and to consider in our blood pressure study only those cases which had, either at the examination or in the past, a finding of high blood pressure and no other impairment whatsoever.

Extent of Study

Because of the amount of detail work involved, we limited ourselves to applicants forty years of age or more with an average systolic of 140 or over and an average diastolic of 90 or over. We were impressed by the number of cases which were found to have some other impairment, past or present, which took them out of the blood pressure group, as shown below:

Number of Cases

	With Blood Pressure Alone	With Other Impairments	Total
Standard	2728	681	3409
Substandard	1606	716	2322
All	4334	1397 – 24.4%	5731

It will be noticed that practically one-fourth of these cases had some evidence of involvement of other parts of the cardiovascular-renal apparatus or other disease. We decided to confine our medical study to the blood pressure cases, though, as will be seen later, we asked the actuaries to study as a separate group those with blood pressure and other impairments. Our plan was to subdivide the blood pressure cases to see what we could find

out about them medically and then ask our Actuarial Department to study as many of our subdivisions as were large enough for accurate mortality studies. In the Medical Department we studied only those cases which could be followed for the whole period under consideration, i.e., issues 1923 to 1928 followed up to March, 1934. That meant leaving out of our calculations all lapsed cases, but as the actuaries were to include the lapsed cases in their studies, we felt that there would be sufficient check on our work when comparing results to offset this factor. As the lapse ratio in all our substandard groups was a constant figure and the lapse ratio in our standard data was also nearly constant, though lower, what we were doing amounted to a study of approximately two-thirds of our substandard material and three-quarters of our standard material. We felt that the results of these studies would have been essentially the same if we had been able to follow up and include the lapsed cases also.

Borderline Group

Since there are differences of opinion about blood pressure, it is to be expected that most of the discussion has to do with the point where blood pressure ceases to be normal and begins to be abnormally high. Everyone admits, for instance, that a systolic of 130 at age forty is normal and that 165 is unduly high, but opinions differ about a systolic of 140 or 145 or even 148; similarly with a diastolic between 90 and 100. On the one hand there are those who believe the M.I.S. experience should be closely followed and that these cases should be given a substantial rating. On the other hand there are those whose experience apparently justifies them in accepting such borderline cases at standard rates. Prior to the blood pressure investigation of 1925 and the Medical Impairment Study, we had in our Company generally accepted as standard risks those whose blood pressure average for both systolic and diastolic was below 150/100. It was decided to study a group of these from our 1923 issues to see what our experience for ten years had been. We wanted particularly to know what our experience had been in cases that had not had at any time a systolic as high as 150

or a diastolic as high as 100 and, of course, had no other impairments, past or present. As our material covered only cases aged forty and over with systolic 140+ and diastolic 90+, this meant we studied a group of individuals aged forty and above who had, in 1923, a systolic between 140 and 149 and a diastolic of 90 to 99 and followed them for a ten-year period. In going over this material, it was found, of course, that standard insurance had been given in 1923 to some whose initial blood pressure on examination was over 150/100, but where subsequent lower readings had brought the average systolic blood pressure below 150 and the average diastolic below 100 mm. There were also cases in this standard group that had a history of blood pressure higher than 150/100, but where the blood pressure on examination in 1923 was within what was then considered to be normal limits.

The experience with these issues of 1923 that had not had a systolic reading as high as 150 or a diastolic as high as 100 is shown in Table III.

The increase in relative mortality with duration is a characteristic which we have not observed in previously published blood pressure data on standard plans, although it did appear in the substandard material of the M.I.S. The table shows that the relative mortality was worst at ages forty to forty-nine, and distinctly better thereafter. As a matter of interest the material was divided so as to separate the cases issued in one large city (about one-third of the data); these showed a mortality ratio of 130% as against 117% for the balance of the material. This class was approached as a borderline class and the mortality results confirm that opinion. In view of the improvement in the examination and the selection of blood pressure cases during the past ten years, it would seem reasonable to assume that with careful underwriting the best of this group of borderline risks might be considered on a standard basis, *provided that all other factors were favorable.*

Highest Blood Pressure

Having eliminated the complex impairment group and having studied those with the blood pressure below 150/100, we pro-

ceeded to study the rest of the material, which, of course, consisted mainly of our substandard issues with a few standard issues which did not meet the requirements of the first group studied. The substandard material was classified according to the highest systolic recorded at any time and also according to the highest diastolic recorded at any time, with results as indicated in Table IV and Table V.

It would appear from this that the mortality increases with considerable regularity as the systolic rises. In this connection reference may be made to an article by Millett who reported a study of 360 cases of hypertension followed from July, 1922, to July, 1932. All these cases had a systolic of 160 or over and 80% had a systolic of 180 or over *at some time* during the period of observation. At the end of this ten-year period 83 or 23% were dead. Blackford and Wilkinson reported 202 patients followed for an average of eight years, at the end of which time 50% were dead, an exceedingly high mortality. Their group covered patients with a systolic of 175 or more and a diastolic of 100 or more.

Diastolic Blood Pressure

The diastolic results are not so uniform as in the case of the systolic, but it must be remembered that these diastolics were taken during a period when there was some doubt as to (a) the ability of all examiners to take diastolics correctly and (b) the point at which the diastolic should be read. Indeed this latter situation still exists. Some companies ask for the diastolic at the change of sound, others when the sound ceases. It is our impression that many physicians who have entered practice since the World War, trained in first-class medical schools, are taking the diastolic as they had been taught in medical schools and are inclined to take it at that phase, irrespective of any instructions given on the insurance blank. It is probable that every company has in its records a mixture of diastolics, some taken at the change of sound, others at the disappearance of sound, although the officials of the company may be led to believe they were all taken at the change of sound or all taken at the disappearance of

sound because the examination blank or instructions call for them at that phase. Incidentally, at the annual meeting in 1921, Dr. Cook, for the Blood Pressure Committee, made the following recommendations:

> "At the Thirty-second Annual Meeting of the Association of Life Insurance Medical Directors of America, October 20th and 21st, 1921, Dr. H. W. Cook presented a report of the Blood Pressure Committee, analyzing the replies received from eleven prominent clinicians, of whom nine were strongly of the opinion that the change in sound at the end of the third phase should be taken to represent the diastolic or minimum pressure. 'From the opinion of the leading internists, it would therefore appear that not only does the end of the third phase represent the true diastolic pressure, but that this is the point which is being taught today by the majority of the better teachers of medicine; and further, that the end of the fourth phase may be very much lower than the true diastolic pressure and therefore very misleading.' The following recommendations were made:
>
> 1st. That the Medical Directors' Association recognize the last loud tone at the end of the third phase as representing the correct diastolic pressure and so instruct their examiners, and that those companies which have trained their examiners to report the end of the fourth phase, or the cessation of all sound, or which feel that this phase gives a valuable check on the examiners, should request both phases.
>
> 2nd. That in those blanks in which both phases are not requested, it be specifically stated in small type under the line for indicating diastolic pressure that the figure given represents the last loud sound at the end of the third phase."

At that time the report was accepted and the matter of carrying out the recommendations referred to the Executive Council. At the meeting of the Executive Council in November, 1922, the recommendations of the Blood Pressure Committee were considered and laid on the table—where apparently they have been ever since! It may be noted that nowadays we talk of the beginning of the fourth and fifth phases, whereas in 1921-22 we

were discussing the end of the third and fourth phases. Even so it would seem that the change of sound was considered at that time the correct diastolic phase. It is our impression that any study of diastolics should be interpreted with caution until all doubt about the phase used has been removed. This is particularly important in view of the fact that a variation of less than five points in the diastolic may affect the numerical rating considerably. It is hoped that arrangements will shortly be made for blood pressure reports to indicate whether the diastolic was taken (a) at the change of sound, (b) the disappearance of sound, or finally if the phase was not known.

Our present examination blank provides for reporting either the fourth or the fifth phase. We have investigated the reports of a number of our examiners and find that in general nearly 50% record the diastolic at the fourth phase and slightly over 50% of this group at the fifth phase, the younger men being more likely to record the fourth phase. As opinions are not unanimous about the difference between the fourth and fifth phases, we made a study of 1,030 cases where both phases were given. Most of these blood pressure readings were obtained through our Home Office examiners, though some of them came from competent examiners in other parts of the country. We found that the average difference between the fourth and fifth phases in these 1,030 cases was 4.98 points, the actual variations being shown in Table VI. It would be interesting to know what other companies have found in this connection.

Synthetic Blood Pressure

It may be inferred from Table IV and Table V that the relative mortality was not disproportionate to the highest systolic and the highest diastolic. It was suggested that there might be some criticism of taking the *one* highest systolic and the *one* highest diastolic as a basis for estimating insurability, as occasionally an individual might show an elevated blood pressure from some temporary disturbance and he ought not to be penalized permanently for this one reading. It was decided, therefore, to take into account the *two* highest readings in cases where more

than one blood pressure was on record. In cases where there were a number of blood pressure reports, the highest systolic and the highest diastolic did not always occur in the same blood pressure reading. We decided to classify the cases according to an arbitrary synthetic blood pressure, formed as follows:

(1) The synthetic systolic was formed by averaging the two highest systolics recorded.
(2) The synthetic diastolic was formed by averaging the two highest diastolics recorded.
(3) The resultant synthetic systolic and synthetic diastolic were used to group the cases in a similar manner to the highest systolic and the highest diastolic (Table V and Table VI). These results are shown in Table VII.

In general the percentage dead rises with the synthetic blood pressure. We felt satisfied, therefore, from these preliminary studies that the highest blood pressure readings were more important in regard to longevity, irrespective of what lower readings might be found at other times. So our Actuarial Department was asked to make a mortality study of as many of the above groups as were large enough to justify it. As stated before, our medical survey includes only existing and dead cases, whereas the actuarial study includes also lapsed cases. A mortality study was also made of the complex impairment group. The results of these studies for standard and for substandard cases are given in Table VIII and Table IX. For the purpose of comparison, the M.I.S. results are given in Table X.

It is interesting to compare the result of this study with the M.I.S. and to realize the different methods pursued in the two studies. In the M.I.S. the material was classified according to the average of the systolic blood pressures found at the time of the medical examination for the policy concerned. In the present New York Life study all factors which had a bearing on the case were considered and the cases were classified according to the average of the two highest systolics and the average of the two highest diastolics reported at any time. While it may be only a coincidence, it is interesting to note the striking parallel

between the mortality results of the two studies, as given in Table X.

Summary

In general it would seem fair to say, as far as the New York Life's contribution is concerned, that the blood pressure cases in the M.I.S. at ages forty and over may be subdivided into three groups.

Group 1. Cases where blood pressure is the only impairment and where the blood pressure has never been 150/100 or higher. The best of this borderline group may possibly be considered on a standard basis.

Group 2. Cases with blood pressure the only impairment but where the blood pressure has at some time been 150/100 or higher. These could be rated according to a synthetic blood pressure formed by averaging the two highest systolics and the two highest diastolics to fit the mortality experience of the M.I.S. substandard groups, 5-15 mm., 16-24 mm., 25-34 mm. and 35-44 mm. above the average systolic.

Group 3. Cases where blood pressure is not the only impairment but where at some time in the past or on the examination at issue some other significant impairment was recorded. If such cases are insured at all, it should be realized that the mortality will probably be approximately 300% of the standard. Further study of this group may possibly be made later to see if any of the accompanying impairments are more important than others, but so far the material is so small that subdivision according to the accompanying impairments as well as the blood pressure would result in groups too small to give reliable results.

Pulse Pressure Analysis

A study of over 1,000 of these synthetic cases, from the standpoint of pulse pressure alone, gave no evidence that the pulse

pressure of itself was important. The largest number of deaths occurred where the pulse pressure was below 40 or over 80. In the former, the diastolics were high and in the latter, the systolics. So the pulse pressure itself could not be said to be the cause of the mortality.

Causes of Death

As a matter of interest, the causes of death in the cases which had become claims were also studied. In 189,609 standard cases studied for the mean blood pressure, there were 5,183 deaths, of which 1,434 or 28% were from cardiovascular-renal disease, these 1,434 being subdivided, as follows:

Heart	801	or 15%
Apoplexy, embolism and thrombosis	398	8%
Renal	235	5%

In the 6,302 substandard cases studied for the mean blood pressure there were 333 deaths, of which 232 or 70% were from cardiovascular-renal disease, these 232 being subdivided, as follows:

Heart	125	or 38%
Apoplexy, embolism and thrombosis	77	23%
Renal	30	9%

It is interesting to note that in the standard group only 28% died of cardiovascular-renal disease, whereas in the substandard group the percentage was 70%.

In the standard and substandard cases studied according to the synthetic blood pressure there were 565 deaths, of which 392 or 69% were due to cardiovascular-renal disease, as follows:

Heart	228	or 40%
Apoplexy, embolism and thrombosis	115	20%
Renal	49	9%

A medical survey of the death certificates of all the claims in the synthetic blood pressure group, including groups too small for actuarial study and carried up to March, 1934, for all the

material, showed 613 deaths, of which 72% were due to cardiovascular-renal disease. In connection with these figures, an article by Murphy, Grill, Pessin and Moxon showed the result of autopsies in 375 cases of hypertensive disease, as follows:

Heart	50%
Renal	10.4%
Apoplexy, embolism and thrombosis	13.4%

with a total of 73.8% dead of cardiovascular-renal disease.

Paullin states in a paper on prognosis in regard to high blood pressure that 15 to 25% died of other diseases, indicating that 75 to 85% died of cardiovascular-renal disease.

It is not always easy when studying the modern death certificate to say whether the immediate cause of death was cardiac, vascular or renal. Many certificates give all three or use the general term cardiovascular-renal disease. In any case, from the death certificates and autopsy reports it is evident that about 70% of these hypertension cases died of cardiovascular-renal disease and nearly one-half of the total number of deaths are due to the heart itself giving out. Stieglitz states that as most deaths in hypertensive cases occur from heart disease, the most important thing in prognosis is *the evaluation of cardiac reserve.* In his opinion the electrocardiogram is not much good for this purpose as there are no changes until there is far-advanced myocarditis, at which time the condition can be noted clinically without an electrocardiogram. The main things to look for are undue dyspnea, tachycardia and cardiac consciousness on exertion. He believes, as do others, that the prognosis is worse if the hypertension occurs at an earlier age and this agrees with the experience of life insurance companies as brought out in the M.I.S. and this study, indicating that the relative mortality is worse in the forties than at older ages.

Estimation of Insurability

The prognosis in hypertension is what we are interested in for underwriting purposes. High blood pressure is but one phase of a syndrome known to physicians as cardiovascular-renal disease

and, in lay insurance circles, as the degenerative diseases. It is part of the process of wear and tear and, as Riesman points out, the stress and strain of life are very potent factors. Overexertion from too much work with too little rest causes the body to wear out too soon, with the result that there are many claims in connection with people who have died in their forties or fifties, when by more careful living they might have lived to be sixty or seventy. Possibly the period which began at about the time of the World War, characterized by high pressure living, was responsible for many people burning themselves up prematurely. It may be that with more leisure and less strenuous living, the claims for degenerative diseases in early middle life will decrease.

Instead of thinking of the high blood pressure as the impairment, we should accustom ourselves to speaking of hypertensive disease. We will then be more likely to remember that this condition is a complex one involving other factors besides the pressure of the blood as revealed by the sphygmomanometer, and that due appreciation of the pathology underlying the disease is necessary to estimate insurability. The whole cardiovascular-renal system must be considered. The heart especially should be carefully examined, not only for murmurs, but for anything which suggests cardiac disease. Incidentally, it is worth remembering that many so-called "functional" murmurs in middle life are associated with hypertensive disease.

In regard to the blood pressure itself, it is suggested that the highest readings are more important. Low readings should not be allowed to minimize the effect of previous high ones. It may be of interest here to quote what Baker, of Rochester, has to say about the effect of exercise on blood pressure. He mentions five groups, as follows:

(a) Normal group where the systolic rises moderately and the diastolic lessens slightly on moderate exertion. The blood pressure returns to normal in three to five minutes.

(b) The initial systolic is higher than in group A, but not definitely abnormal. The systolic rises more precipitately, the diastolic remaining the same or rising slightly. The blood pressure

returns to normal in five minutes or less after exercise. This group includes the middle-aged from forty-five to fifty showing a normal reaction to beginning age.

(c) Systolic definitely hypertensive on the initial reading. Exercise causes the systolic to rise abruptly to a higher level and the diastolic rises higher than in group (b). Both take much longer to return to the original reading.

(d) Fixed hypertension with dyspnea on the mildest exertion. Exercise is contra-indicated in this group.

(e) Systolic and diastolic fall markedly on exercise and do not return to normal in ten to fifteen minutes. This is a dangerous group because other changes, such as myocarditis, have already occurred.

Finally, evidence of past or present disease of other organs should not be overlooked and due consideration should be given to the individual's build, occupation, social status and mode of living.

ACKNOWLEDGMENT

While the number of substandard cases involved in this study is small compared to the M.I.S., yet a careful review of the original papers involved a large amount of detail work, which would not have been possible without the assistance of Dr. R. A. Fraser, Chief Medical Director, and my associates, Dr. F. C. Evers, Dr. W. D. Heaton, Dr. I. C. Lawler, Dr. E. M. Freeland and Dr. R. W. Hipsley. Our Actuarial Department, under Dr. Hunter's guidance, has been, as usual, extremely helpful. Mr. Bowerman especially has given considerable time to the study and it is largely due to his efforts that we are able to present the tabular data. Whatever merit this study may have is entirely due to the active co-operation of many workers rather than to the efforts of any one individual.

TABLE I

Blood Pressure Investigation—by "Mean" of Systolic and Diastolic Blood Pressures. New York Life.

Exposures Carried to Anniversary in 1932. Expected Deaths by Company's Standard. Standard Issues of March, 1923, to March, 1924, inclusive.

"Mean" Blood Pressure Group	Deviation in Mm. from "Mean"	No. of Lives Dead	No. of Policies Dead	Expected Dead	Policies Ratio	No. of Entrants	Average Duration
Low	−16 to −24	24	27	28.94	93%	1121	4.5
Underaverage	− 5 to −15	1131	1326	1452.22	91	53112	4.5
Average	− 4 to + 4	2056	2368	2432.09	97	97026	4.5
Overaverage	+ 5 to +15	1168	1408	1210.30	116	37517	4.5
High	+16 to +24	44	54	35.82	151	833	4.5
All		4423	5183	5159.37	100%	189609	4.5

	LOW			UNDERAVERAGE			AVERAGE			OVERAVERAGE			HIGH		
	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio
By Duration															
1– 5	11	15.31	72%	672	769.22	87%	1277	1301.39	98%	677	630.81	107%	24	18.23	132%
6–10	16	13.63	117	654	683.00	96	1091	1130.70	96	731	579.49	126	30	17.59	171
All	27	28.94	93%	1326	1452.22	91%	2368	2432.09	97%	1408	1210.30	116%	54	35.82	151%
By Age at Entry															
15–39	16	16.73	96%	727	755.92	96%	1346	1330.43	101%	455	441.48	103%	11	7.42	148%
40–49	5	6.04	83*	330	388.06	85	605	646.59	94	553	453.79	122	30	17.93	167
50–65	6	6.17	97*	269	308.24	87	417	455.07	92	400	315.03	127	13	10.47	124
All	27	28.94	93%	1326	1452.22	91%	2368	2432.09	97%	1408	1210.30	116%	54	35.82	151%

*As there were so few deaths in this group the results should be interpreted with caution.

TABLE II

Blood Pressure Investigation—by "Mean" of Systolic and Diastolic Blood Pressures.

New York Life.

Exposures Carried to Anniversary in 1932. Expected Deaths by Company's Standard.

Issues of 1925-1931, inclusive, Substandard on Account of Abnormal Blood Pressure.

"Mean" Blood Pressure Group	Deviation in Mm. from "Mean"	No. of Lives Dead	No. of Policies Dead	Expected Dead	Policies Ratio	No. of Entrants	Average Duration
Overaverage	+ 5 to +15	93	116	66.81	174%	2442	2.5
High	+16 to +24	136	177	73.98	239	3160	2.6
Higher	+25 to +35	33	40	12.88	311	700	2.7
All		262	333	153.67	217%	6302	2.6

	OVERAVERAGE			HIGH			HIGHER		
	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio	Actual Dead	Expected Dead	Ratio
By Duration									
1– 5	112	61.42	182%	156	65.54	238%	31	11.32	274%
6– 7	4	5.39	74*	21	8.44	249	9	1.56	577
All	116	66.81	174%	177	73.98	239%	40	12.88	311%
By Age at Entry									
15–39	5	4.21	119%*	22	6.57	335%	13	1.80	722%
40–49	18	8.96	201	71	23.94	297	16	6.90	232
50–65	93	53.64	173	84	43.47	193	11	4.18	263
All	116	66.81	174%	177	73.98	239%	40	12.88	311%

NEW YORK LIFE 1925-1931		M.I.S. 1909-1927	
Deviation from "Mean" Blood Pressure in Mm.	Mortality Ratio	Deviation from Average Systolic Blood Pressure in Mm.	Mortality Ratio
+ 5 to +15	174%	+ 5 to +15	153%
+16 to +24	239%	+16 to +24	181%
+25 to +35	311%	+25 to +35	205%

*As there were so few deaths in this group the results should be interpreted with caution.

TABLE III

Systolic 140-149 Mm. and Diastolic 90-99 Mm.
Exposures Carried to Anniversary in 1932.
Standard Issues of March, 1923, to March, 1924, Inclusive.

	No. of Lives Dead	No. of Policies Dead	Expected* Dead	Ratio	No. of Entrants	Average Duration
Age 40 and over	177	217	178.84	121%	2086	4.7 years

*Expected deaths are by the Company's standard.

By Duration	Actual Dead	Expected Dead	Ratio	By Age at Entry	Actual Dead	Expected Dead	Ratio
1– 5	81	87.70	92%	40–49	96	67.24	144%
6–10	136	91.14	149	50–59	104	91.50	113
				60 and over	17	20.10	85
All	217	178.84	121%	All	217	178.84	121%

TABLE IV

Highest Systolic, Substandard Issues 1923-1928, Inclusive.
Existing and Dead (Excluding Lapsed) Age 40 and Over.

Systolic Blood Pressure	Living	Dead	Total	Per Cent. Dead	Graded Per Cent. Dead
1. 140–144	15	1	16	6	9
2. 145–149	15	3	18	17	12
3. 150–154	124	22	146	15	15
4. 155–159	143	32	175	18	18
5. 160–164	265	73	338	22	21
6. 165–169	87	26	113	23	23
7. 170–174	93	28	121	23	27
8. 175–179	19	8	27	30	30
9. 180–184	23	13	36	36	33
10. 185+	22	12	34	35	36
	806	218	1024		

TABLE V

Highest Diastolic, Substandard Issues 1923-1928, Inclusive. Existing and Dead (Excluding Lapsed) Age 40 and Over.

Diastolic Blood Pressure	Living	Dead	Total	Per Cent. Dead	Graded Per Cent. Dead
1. 90– 94	161	38	199	19	17
2. 95– 99	108	19	127	15	18
3. 100–104	316	71	387	18	20
4. 105–109	76	34	110	31	23
5. 110–114	113	28	141	19	26
6. 115–119	19	7	26	27	31
7. 120–124	13	11	24	46	37
8. 125+	6	4	10	40	43
	812	212	1024		

TABLE VI

Difference in Mms. Between 4th and 5th Phase Diastolics. Age 10 to 69, Systolic 102-220, Diastolic (4th Phase) 52-120.

Number of Cases	Millimeters
106	0
0	1
177	2
7	3
317	4
41	5
173	6
7	7
74	8
4	9
80	10
1	11
14	12
1	13
11	14
3	15
3	16
0	17
2	18
0	19
8	20
0	21
1	22
1030	Average Difference 4.98

TABLE VII

Synthetic Blood Pressure, Substandard and Remainder of Standard Issues 1923-1928, Inclusive.

Existing and Dead (Excluding Lapsed) Age 40 and Over.

	Blood Pressure Systolic	Blood Pressure Diastolic	Living	Dead	Total	Per Cent. Dead	Graded Per Cent. Dead
1.	135–139	92– 94	1	0	1	0	6
2.	140–144	95– 97	14	3	17	18	12
3.	145–149	98– 99	68	18	86	21	17
4.	150–154	100–101	398	84	482	17	19
5.	155–159	102–103	246	63	309	20	21
6.	160–164	104	185	53	238	22	24
7.	165–169	105	105	40	145	28	28
8.	170+	106	172	84	256	33	32
			1189	345	1534		

TABLE VIII

Blood Pressure Investigation—Group 4 and Blood Pressure With Other Impairments.

Exposures Carried to Anniversary in 1933. Expected Deaths by Company's Standard.

Standard Issues of 1923 and 1924, Age 40 and Over.

	Systolic or Diastolic Synthetic Blood Pressure	No. of Lives Dead	No. of Policies Dead	Expected Dead	Ratio	No. of Entrants	Average Duration
Blood Pressure Group 4	150–154 / 100–101	55	69	52.29	132%	427	5.0
Blood Pressure With Other Impairments		112	138	79.89	173%	805	4.9

	BY POLICY YEAR				BY AGE AT ISSUE			
	Policy Year	Actual Dead	Expected Dead	Ratio	Age at Issue	Actual Dead	Expected Dead	Ratio
Blood Pressure Group 4	1– 5	30	21.65	139%	40–49	25	11.72	213%
	6–10	39	30.64	127	50 & over	44	40.57	108
	All	69	52.29	132%	All	69	52.29	132%
Blood Pressure With Other Impairments	1– 5	44	35.25	125%	40–49	49	25.58	192%
	6–10	94	44.65	211	50 & over	89	54.32	164
	All	138	79.90	173%	All	138	79.90	173%

TABLE IX

Blood Pressure Investigation—By Synthetic Systolic or Diastolic Blood Pressure, i.e., By the Average of the Two Highest Systolic and Two Highest Diastolic Pressures.

Exposures Carried to Anniversary in 1933. Expected Deaths by Company's Standard. Substandard Issues of 1923-1928, inclusive, Age 40 and Over.

Blood Pressure Group	Systolic or Diastolic Synthetic Blood Pressure	No. of Lives Dead	No. of Policies Dead	Expected Dead	Ratio	No. of Entrants	Average Duration
4	150–154 / 100–101	13	14	19.55	72%	284	3.8
5	155–159 / 102–103	39	48	33.24	144	461	3.9
6	160–164 / 104	39	48	25.87	186	371	3.8
7	165–169 / 105	31	35	14.85	236	231	3.8
8	170–174 / 106+	55	70	28.07	249	431	3.8
All		177	215	121.58	177%	1778	3.9
Blood Pressure with Other Impairments		116	143	45.51	314%	828	3.6

Synthetic Blood Pressure Group	BY POLICY YEAR Policy Year	Actual Dead	Expected Dead	Ratio	BY AGE AT ISSUE Age at Issue	Actual Dead	Expected Dead	Ratio
4	1–5	10	12.22	82%*	40–49	5	6.80	74%*
	6–10	4	7.33	55 *	50 & over	9	12.75	71 *
	All	14	19.55	72%	All	14	19.55	72%
5	1–5	28	20.99	133%	40–49	21	10.04	209%
	6–10	20	12.25	163	50 & over	27	23.20	116
	All	48	33.24	144%	All	48	33.24	144%
6	1–5	32	16.35	196%	40–49	18	6.71	268%
	6–10	16	9.52	168	50 & over	30	19.16	157
	All	48	25.87	186%	All	48	25.87	186%
7	1–5	24	9.51	252%	40–49	12	5.05	238%
	6–10	11	5.34	206	50 & over	23	9.80	235
	All	35	14.85	236%	All	35	14.85	236%
8	1–5	46	17.15	268%	40–49	32	8.97	357%
	6–10	24	10.92	220	50 & over	38	19.10	199
	All	70	28.07	249%	All	70	28.07	249%
Blood Pressure with Other Impairments								
	1–5	108	31.55	342%	40–49	60	15.72	382%
	6–10	35	13.96	251	50 & over	83	29.79	279
	All	143	45.51	314%	All	143	45.51	314%

*As there were so few deaths in this group the results should be interpreted with caution.

TABLE X

Comparison of M.I.S. and New York Life Insurance Company (1934).

M.I.S. 1909-1927 ISSUES		N. Y. L. I. CO. (1934) 1923-1928 ISSUES	
Blood Pressure	Mortality Ratio Age 40 and Over	Blood Pressure	Mortality Ratio Age 40 and Over
5–15 Mm. Overaverage	161%	155–159 / 102–103	144%
16–24	187%	160–164 / 104	186%
25–34	212%	165–169 / 105	236%
35–44	253%	170–174 / 106	249%

Detail Tables.

M.I.S. 5-15 MM. ABOVE AVERAGE SYSTOLIC — **N. Y. L. I. CO. (1934)—155-159 / 102-103**

BY POLICY YEAR

Year	Ratio, Actual to Expected		Year	Ratio, Actual to Expected
1–2	137%	151%	1–5	133%
3–5	162		6–10	163
6–10	159			
11–19	169			
All	153%		All	144%

BY AGE AT ISSUE

Age	Ratio, Actual to Expected		Age	Ratio, Actual to Expected
15–29	105%		40–49	209%
30–39	129		50+	116
40–49	178	161%		
50+	150			
All	153%		All	144%

M.I.S. 16-24 MM. ABOVE AVERAGE SYSTOLIC — **N. Y. L. I. CO. (1934)—160-164 / 104**

BY POLICY YEAR

Year	Ratio, Actual to Expected		Year	Ratio, Actual to Expected
1–2	143%	160%	1–5	196%
3–5	196		6–10	168
6–10	244			
11–19	173			
All	181%		All	186%

TABLE X—Continued

M.I.S. 16-24 MM. ABOVE AVERAGE SYSTOLIC — N. Y. L. I. CO. (1934)—160-164/104

BY AGE AT ISSUE

Age	Ratio, Actual to Expected		Age	Ratio, Actual to Expected
15–29	85%		40–49	268%
30–39	175		50+	157
40–49	245	187%		
50+	162			
All	181%		All	186%

M.I.S. 25-34 MM. ABOVE AVERAGE SYSTOLIC — N. Y. L. I. CO. (1934)—165-169/105

BY POLICY YEAR

Year	Ratio, Actual to Expected		Year	Ratio, Actual to Expected
1–2	177%	196%	1–5	252%
3–5	213		6–10	206
6–10	219			
11–19	230			
All	205%		All	236%

BY AGE AT ISSUE

Age	Ratio, Actual to Expected		Age	Ratio, Actual to Expected
15–29	75%		40–49	238%
30–39	221		50+	235
40–49	253	212%		
50+	187			
All	205%		All	236%

M.I.S. 35-44 MM. ABOVE AVERAGE SYSTOLIC — N. Y. L. I. CO. (1934)—170-174/106

BY POLICY YEAR

Year	Ratio, Actual to Expected		Year	Ratio, Actual to Expected
1–2	210%	237%	1–5	268%
3–5	260		6–10	220
6–10	353			
11–19	197			
All	265%		All	249%

BY AGE AT ISSUE

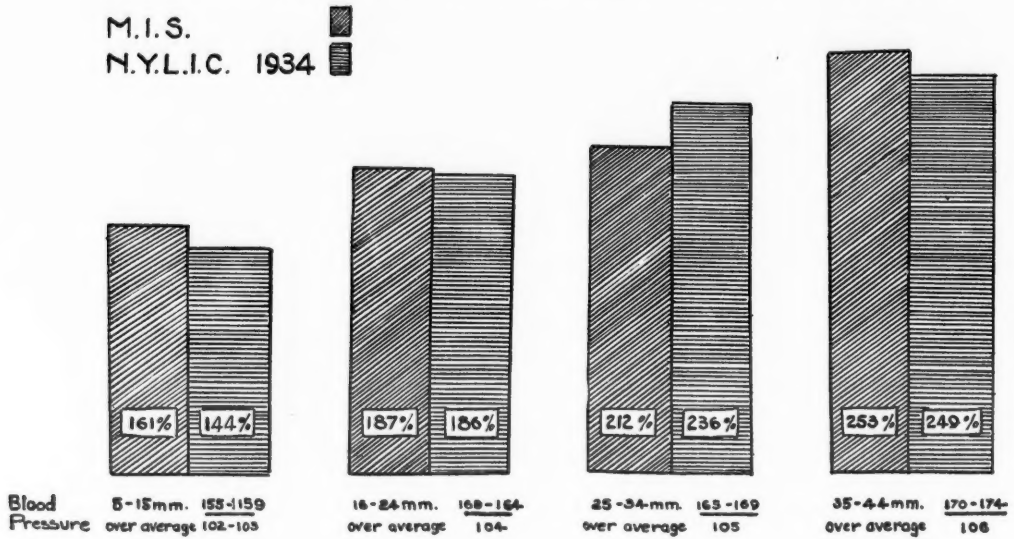
Age	Ratio, Actual to Expected		Age	Ratio, Actual to Expected
15–29	154%		40–49	357%
30–39	383		50+	199
40–49	304	253%		
50+	195			
All	265%		All	249%

NEW YORK LIFE INSURANCE COMPANY

Blood Pressure Investigation 1934

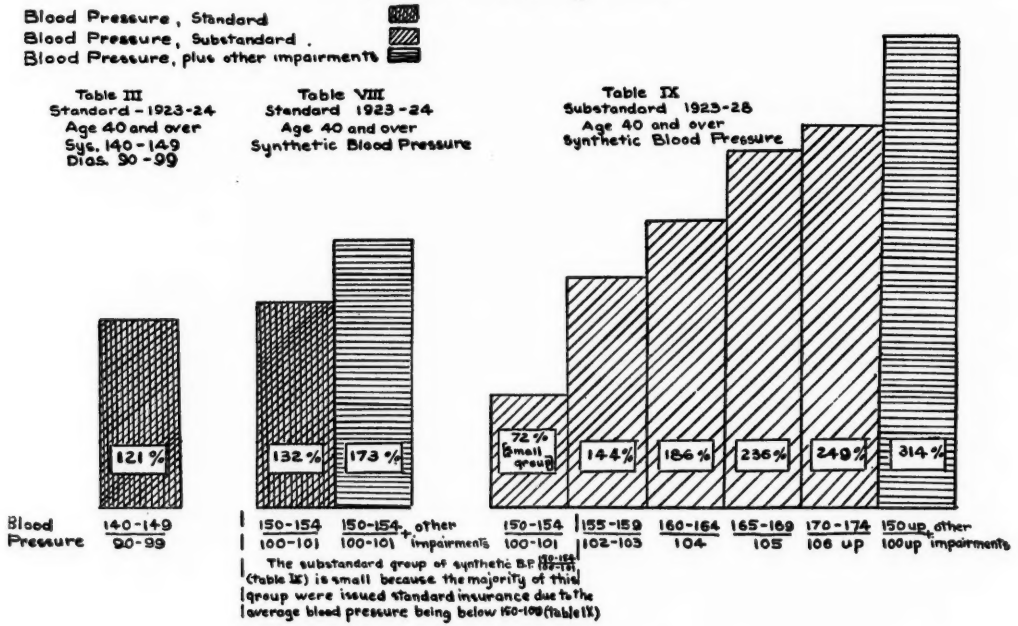
Table IX

Mortality Rates of M.I.S. and N.Y.L.I.C. 1934



NEW YORK LIFE INSURANCE COMPANY

Blood Pressure Investigation 1934
Comparative Mortality Ratios



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DR. FRASER—The discussion of this paper will be undertaken first by Dr. S. J. Streight, Medical Director of the Canada Life Assurance Company. Dr. Streight. In the absence of Dr. Streight, Dr. Scadding will act for him.

DR. SCADDING—(Read Dr. Streight's paper.)

I have read and re-read Dr. Bolt's paper and can assure those of you who have not done so that it will well repay you for the time and effort required to give it careful study. This essay on "Hypertension, An Underwriting Problem" is so full of thought-provoking material that I find it difficult to discuss it without going farther afield than one feels is permissible in a discussion. Not having any comparable study to present, my remarks must of necessity be based upon clinical observation and theoretical consideration of the factors which may be responsible for the unfavorable mortality which has been shown to result where

there is a sustained, even though not a marked, departure from normal.

Relating to the Cause and Development

The living body is a well equipped laboratory in which Nature is constantly working out experiments. Some of these experiments, like those in any laboratory, are crowned with success while others fail. When Nature's experiments succeed, our bodily functions proceed normally and we are not aware that anything is happening and we are in a state of good health. When these experiments fail, because of some disturbance of the normal nervous or chemical balance, we may be conscious of unpleasant sensations, which we have learned to recognize as symptoms of disease.

Health is maintained by reason of a constant, purposeful effort to preserve physiological equilibrium in the reactions constantly proceeding between the cells of the body and their nutrient capillaries. Changes of a physio-chemical character, which originate in the capillary system are, in all probability, the first evidence of pathological change, or disease. Since all metabolic changes take place through the capillary walls, they are naturally the most vulnerable, as well as the most active and purposive, part of the vascular system. The capillary system is affected by both chemical and vasomotor stimuli, the former as a coarse and the latter as a fine adjustment.

The capillaries form a constituent part of the tissues in which they lie, and the circulation of blood through them is regulated and controlled by the requirements of the individual cells of the structures they supply. The vital processes of life are dependent upon the state of the capillary system and particularly upon the condition of the walls of these minute vessels. The health of every organ and cell depends upon the quality and the quantity of the blood circulating in its capillary system.

We can readily understand how chemical change, the result of repeated toxic processes, may so alter contractility as to produce, at first, intermittent hypertonicity of the capillaries and arterioles, giving us the earliest evidence of abnormal blood pressure re-

action. Later, if these chemical irritants continue to act, we have continuous hypertonicity, and still later, degeneration of the musculature of the arterioles, capillaries, and arteries, and the entire vascular system. Too often we fail to realize that these early departures from normal may be the first evidence of beginning hypertension.

On Reading Blood Pressure

Brief comment upon the actual taking of blood pressure may not be out of order. The cuff should be carefully adjusted to the arm and gently inflated. It is advisable to first observe the diastolic pressure. This, besides being re-assuring to the applicant, enables the examiner, I believe, to obtain a more accurate observation of the actual diastolic pressure and before it is changed by the possible disturbing effect of any extraneous influence. To observe the systolic pressure, the cuff should be inflated to carry the column of mercury well above any anticipated reading. Pressure should then be gradually released. The first sound to appear will indicate the systolic pressure. At the same time the finger should be kept on the radial pulse so that the examiner may detect a silent phase, if such exists. Pressures, both systolic and diastolic, should be read with the mercury falling, and the period of inflation should be as brief as possible. We should not overlook the fact that physicians in general, including examiners, are inclined, when the pressure is variable, to record the more favourable observations.

Systolic pressure represents the maximum intra-arterial pressure at the end of cardiac systole. It therefore gives some indication of the amount of blood expelled into the arterial tree at each systole plus the peripheral resistance. The diastolic pressure represents the intra-arterial pressure during the diastolic phase of the cardiac cycle. It indicates the minimum pressure to which the arterial wall is constantly subjected and also the initial pressure which the left ventricle has to overcome at the beginning of systole before it can expel its contents into the arterial system. A sustained diastolic pressure places a constant extra burden upon the heart and, to quote David Riesman, "Few hearts can endure long a constantly high diastolic pressure."

Clinical Opinion

C. W. C. Bain, in the British Medical Journal of January, 1932, states that in hyperpiesia there is elevation of both systolic and diastolic; that increase must be sustained, not temporary; that the diastolic pressure is more important that the systolic. He considers that a sustained blood pressure of 150 systolic and 90 diastolic is evidence of hyperpiesia; that hyperpietic crises are likely to occur at any stage with swift, definite elevations of pressure; and that, after a time, the cardio-vascular system responds to the stress of these continued high pressures by structural changes which are permanent. Hypertrophy of the left ventricle develops. Obliterative changes occur in the arterioles. These obliterative changes increase the peripheral resistance, and further increase in blood pressure is necessary to maintain the circulation.

Bramwell and Ellis, in the British Medical Journal of March, 1928, conclude that high pulse pressure in old age is a compensatory mechanism to overcome the loss of elasticity in the arteries. Even apart from any structural change, the elasticity of the arterial walls might be greatly impaired by a high diastolic. Hence a relatively small rise in diastolic pressure would entail a much greater rise in pulse pressure if the heart were to maintain its output. The diastolic pressure is of special significance in relation to the ultimate prognosis because it represents the liability which the heart is called upon to undertake. The systolic pressure, on the other hand, is more closely related to the immediate issue since it indicates the capacity of the heart to meet its liabilities. A falling systolic, associated with a rising diastolic pressure, is always a very bad omen in that it signifies heart failure. The structural changes which were found post-mortem in the earliest cases of essential hypertension were (1) hypertrophy of the left ventricle of the heart, (2) hypertrophy of the middle coats of the arteries (generally accompanied by some fibrosis of the media, a degenerative feature, but an earlier stage of pure hypertrophy might be presumed), (3) changes in the minute arterioles of the size of the afferents to the renal glomeruli.

It is apparent that clinical medicine is slowly but definitely accumulating evidence to show that slight to moderate increase in the systolic or diastolic pressure, or in both, is followed by pathological change. At a recent meeting of the Medical Section of the American Life Convention, Dr. Willius, of the Mayo Clinic, made the significant statement that he considered any sustained systolic pressure of 140 mm. or diastolic of 90 mm. to be evidence of hypertension. Our responsibility is to recognize evidence of the earliest departure from normal, to determine, if possible, the rate at which such change is likely to progress and the probable duration of life before the end results become evident.

New York Life Investigation

It would be interesting to know the actual average systolic and diastolic pressure for the different age distributions from which the mean used in this study was obtained. It would enable us to visualize and more easily compare the Medical Impairment Study with the method of studying by mean pressures. Did the mean, upon which Tables 1 and 2 are based, approximate that of the normal pressures as given in the M.I.S.? If so, the departure of +5 to +15 from the mean, referred to in the Over-Average Group, and +16 to +24, referred to in the High Group in Table 1, Standard Lives, show a marked degree of similarity between the present experience of the New York Life and that of the Medical Impairment Study of 1929. They each appear to substantiate the opinion held by many medical directors and clinicians, namely, that a sustained systolic pressure of 140 mm. or a diastolic pressure of 90 mm. or over is pathological and accompanied by definite increase in the number of deaths from cardio-vascular-renal disease.

Comparison of the mortality resulting from the Over-Average and High Groups, Table 1, accepted on a standard basis, with that of the groups showing similar deviation from normal, Table 2, accepted on substandard basis, demonstrates that a favourable mortality may be obtained where selection is sufficiently rigid. Dr. Bolt remarks, "There must obviously have been some other

factors beside blood pressure which affected the selectors when deciding the ratings."—possibly family history, type of applicant, amount of insurance, age distribution, and any tendency on the part of the examiners to record the most favourable of several readings rather than all the observations made. Did the standard section contain a large percentage of the most favourable end of the group, from the point of view of age and departure from normal bood pressure? Further investigation of this material might clear this point.

Averaging of a number of blood pressures, or accepting the most favourable of several, is not likely to give us exact information. If we are satisfied that an unfavorable reading is accidental or in error, why not disregard it entirely and base our decision upon subsequent observations, rather than confuse the issue and mislead ourselves by averaging a number of records, some of which we feel are unreliable? On the contrary, when we are satisfied that the evidence before us indicates beginning hypertension, we should not hesistate to classify according to the degree of departure from normal.

The difference in mortality between the standard and substandard groups, Tables 1 and 2, is a warning to us how carefully we must study all applicants with hypertension for evidence of organic changes or even the suspicion of such. Change in contour. of the heart, or increase in its rate of action, is highly important.

Our experience, like that of the New York Life, has been that where subsequent readings are requested they are as a rule more favorable to the applicant, except where the insurance history presents an unfavorable record, and sometimes even in those cases. In asking for additional observations, we should be careful that we do not so word our request that the examiner may interpret it as an invitation to report more favorably. We will probably obtain much more exact information if, when we ask our examiner to make a re-check, we stipulate that this be done during the latter part of the day's occupation and under as nearly normal working conditions as possible. If we have reason to suspect that the applicant may be aware of the

condition, care should be taken to see that he is not given an opportunity to prepare himself for further observations. It might be said in favor of obtaining additional readings that this satisfies the applicant and agent that we are fair, and enables us at the same time to obtain additional urinary specimens for examination.

The concensus of clinical opinion appears to be that hypertension requires a considerable period of exposure before the end results are evident. The Medical Impairment Study of 1929 and the present study indicate that the maximum, or peak, mortality is reached between the sixth and tenth years of exposure and that thereafter, while remaining at a sustained high level, is slightly below the maximum. Entrants forty years of age or over may arrive at the peak within a ten year period, but I am not at all convinced that the applicants under age forty will have attained the maximum mortality within ten years of exposure.

From the interest that is being shown in hypertension, it is evident we are all regarding this condition with much greater concern than formerly. This is particularly true regarding a history of previous unfavorable records.

In earlier observations, the examiner no doubt recorded the pressures as first observed, without very much concern or effort to obtain more favorable readings, as neither insurance nor clinical medicine had learned to appreciate the significance of moderate departures from normal. Today, examiners are well aware of the importance of readings in excess of 140/90 and, appreciative of the possible disturbing effect of an examination, may make a distinct effort to place the applicant at ease and to observe the pressures under as favorable circumstances as possible. The result may be distinctly favorable to the applicant.

While Tables 4 and 5 do not state the normal percentage of dead or the age distribution, they appear to indicate that, with any increase in systolic or diastolic, there is a definite increase in mortality.

Variable blood pressure, Dr. Bolt points out, has not received the attention it merits. Blood pressure may show some variation, depending upon circumstances at the time of reading, even

in healthy normals. However, any marked variation must be considered as possible evidence of beginning hypertension. Blood pressure observations in the basal state (i.e., that condition in which no exercise or other disturbing factor has intervened from the sleeping state) are decidedly more favorable than daytime observations under average conditions. Any group having abnormal readings on one or more examinations, and with subsequent observations more favorable or normal, will, I feel sure, show a less favorable experience than a comparable group where such variation does not exist. This applies to all age distributions but is particularly applicable to entrants between thirty-five and fifty years of age (see the Medical Impairment Study, page 40, "At entry ages thirty-five to forty-nine, half of the data appeared with a mortality of 232%.")

Abnormal blood pressure, attributed by the examiner to nervousness, excitement, psychic causes, emotion, worry, anxiety, fatigue, overwork, etcetera, may indeed be evidence that these people are potential hypertensives—at any rate it suggests some vascular irritability. No doubt a follow-up of such a group would show a progressive increase in hypertension in a higher percentage of them than in a similar group not showing such variable findings. Menard examined the blood pressure of French soldiers before and after active combat and during various stanges of trench warfare. The net result was to indicate that prolonged mental and physical stimulation, sufficient to produce fatigue, reduced systolic and diastolic pressure, but the former more than the latter.

Dr. Bolt invites our attention to the point at which the diastolic pressure should be read. Unquestionably, the moment at which the diastolic pressure suddenly changes from a distinct snapping quality to a muffled whiff is the point indicating the true diastolic. This requires careful observation since, should the reading be recorded at the appearance of the mufflled whiff rather than the last clear tone, we may have a reading definitely lower than the actual diastolic. Where the examiner is experienced in reading blood pressures, the observation of the exact moment of

change should not present any difficulty. Our forms should indicate at which point the diastolic is to be observed.

It has been our practice, since blood pressure observations were first required, to ask for the diastolic at the moment of disappearance of all sound. My own observation in practice in making insurance examinations and examination of soldiers during the war has been that the difference between the tone change and the disappearance of all sound averages about five millemetres. Dr. Fisher and Dr. Westrand (Volume XIX of this Association, page 140, Table B) reported a uniform difference of five millemetres in a study of 20,735 lives at ages twenty to sixty.

Presumably, the material in Table 3 was included in Table 1; yet the resulting mortality (121%) of the group "Systolic 140-149 and Diastolic 90-99 mm.", Table 3, is more favorable than that of the "High Group, Plus 16-24", Table 1 (Mortality 151%). While the mortality of the entire group, Table 3, is favorable, that for the age distribution 40-49 is 144%. Does this not indicate that it is dangerous to accept on a standard basis applicants under fifty years of age who come within the classification in Table 3? It would be interesting to follow this group and observe the mortality for durations up to twenty years or longer.

Study by Mean Pressures

All tables extant have given more weight to a variation in diastolic than to an equal variation in systolic. The use of mean pressures gives equal weight to a given variation whether it be diastolic or systolic. If variations in either were always accompanied by proportional variations in the other, the use of mean might be justified, but not otherwise. The evidence submitted does not appear to justify the use of a combined table, based on mean departures, for estimation of mortality.

Summary of Points Brought Out by Dr. Bolt's Paper

The highest readings must not be disregarded.

Favorable findings should not be allowed to minimize the importance of previous unfavorable findings.

Even moderate degrees of hypertension may be accompanied by organic change which it is impossible to diagnose in the early stages.

Change in the size of the heart, particularly change in contour, is important.

The most unfavorable experience for even moderate departures from normal is found in the age group 40-49.

Abnormal diastolic pressure is of greater importance from the standpoint of prognosis than systolic.

Moderate hypertension is much more serious when accompanied by even a moderate acceleration in the rate of the heart's action.

Observations should be made under ordinary daytime working conditions.

In applicants under age fifty, systolic 140 mm. and diastolic 90 mm. should be regarded with suspicion and, if we are to consider on a standard basis, we should be satisfied that no other unfavorable feature exists. Where the systolic is 145 mm. or over or where the diastolic is 95 mm. or over, I believe we are dealing with a class that is definitely substandard, and where accompanied by other impairments, even though not recognizable, the experience is likely to be decidedly unfavorable (see Table 2).

A uniform practice, with regard to the point at which to record diastolic pressure, is needed.

Change in environment, the result of the extension of man's control over physical forces (such as may be seen in the development of speed and the elimination of time and space) no doubt has brought about some change in the character of disease. During this period, have we developed a capacity to adjust ourselves to such rapidly changing conditions? If there is an increase in the incidence of hypertension, does it in some degree reflect our mode of life?

I wish to thank Dr. Bolt for the privilege and pleasure of discussing his paper.

DR. FRASER—The next discusser of this paper was to be Mr. Horace R. Bassford, of the Metropolitan Life Insurance Company. Unfortunately, Mr. Bassford is on the Coast, but he has sent an able representative to bat for him, Mr. Edward A. Lew. Mr. Lew.

MR. LEW—(Read Mr. Bassford's paper.)

MR. BASSFORD—Dr. Bolt is to be heartily congratulated on his original and thought-provoking proposal for underwriting risks with abnormal blood pressure, and on his excellent discussion of some of the leading inferences regarding blood pressure.

Although differing with Dr. Bolt on the interpretation of some of the data presented and somewhat uncertain as to the form of the ratings which are implied by Dr. Bolt's proposal, I find myself in close agreement with what I believe to be the main issues of the paper.

The first of these is that moderate departures from average blood pressure unaccompanied by any medical impairment constitute from the viewpoint of expected mortality a definite medical impairment. The data presented in recent years by Dr. Hunter (1), Dr. Mackenzie (2), Mr. Blackadar (3), the writer (4,5), and now by Dr. Bolt have clearly established the fact that a departure of 20 mm. from average systolic marks a risk as borderline. In the light of these findings it becomes impossible under any circumstances to defend the acceptance for standard insurance of blood pressures much in excess of this borderline. Our company's notion as to the proper limits for standard insurance is embodied in the rating schedule referred to later on, which schedule is under consideration for adoption by the Metropolitan. The ratings appearing in this schedule pertain to various combinations of the averages of all systolic and all diastolic (5th phase) readings taken on an application, including history within two years. The proposed upper limits for standard insurance are summarized in the following table:

TABLE 1

SUGGESTED LIMITS OF BLOOD PRESSURE FOR STANDARD INSURANCE

Age	Systolic / Diastolic (Fifth Phase)
20	140/90
30	142/92
40	145/92
50	148/94

The above limits appear to be in line with Dr. Bolt's recommendations for borderline risks:—those with blood pressure between 140/90 and 149/99 at ages 40 and over, only the best of such risks being recommended for standard, if free of medical impairment and with a history of blood pressure not exceeding 150/100.

The second main point is that for substantial departures from average blood pressure unaccompanied by other medical impairment, the M.I.S. findings for systolic pressure present as good a picture of the mortality to be expected as is available. Dr. Bolt's data on this point are shown in his Table X. Though not directly comparable with the M.I.S. material, they nevertheless can be shown to constitute categorical evidence in support of the aforementioned thesis. We, in the Metropolitan, were led to the same conclusion by noting that with increasing departure from average systolic pressure, the margin between the mortality ratios in our standard lives' experience and the corresponding mortality ratios in the M.I.S. narrowed rapidly.

In presenting his data and in the formulation of his proposals for underwriting risks with abnormal blood pressure, Dr. Bolt has experimented with several indices to represent the blood pressure of individual cases. In turn Dr. Bolt has tried to describe the blood pressure of individual cases by means of (a) the one mean of all the systolic and diastolic readings combined (b) the two averages of all the systolic and all the diastolic readings (as in the M.I.S.) (c) the highest systolic and highest diastolic readings and (d) the averages of the two highest systolic and the two highest diastolic

readings. The problem of finding a satisfactory index of an individual's blood pressure appears to be of such importance as to warrant a most careful scrutiny of the rationale underlying the use of the several indices dealt with by Dr. Bolt.

The use of the one mean of all the systolic and diastolic readings combined is open to the serious criticism of entirely neglecting pulse pressure. A satisfactory index of blood pressure must obviously take account of unfavorable pulse pressure variations. When it is realized that such cases as 135/90 and 160/65 result in an identical "mean" blood pressure, the use of this index will be restricted to cases within the normal range of pulse pressure.

In connection with the use of the "mean" blood pressure it should also be noted that a given departure from average "mean" blood pressure is equivalent to 6/5 the given departure from average systolic pressure, assuming a 3:2 relationship between the systolic and diastolic pressures. For instance, a blood pressure of 149/97 at age 20 represents a departure of +25 mm. from average "mean" pressure and a departure of +29 mm. (or about 6/5 × 25) from average systolic pressure. This is the principal reason why the N. Y. Life's experience on its 1925-1931 substandard issues shows for given departures from average "mean" pressure considerably higher mortality ratios than the M.I.S. experience for corresponding departures from average systolic pressures. Another reason for the M.I.S. blood pressure experience showing lower mortality ratios is that the M.I.S. standard table probably overstates the actual mortality in the years covering the bulk of the blood pressure experience. This would tend to overstate the expected deaths and understate the blood pressure mortality ratios to a small extent.

The use of the two averages of all systolic and all diastolic readings, taken separately, to represent the blood pressure of an individual has been criticized as giving undue weight to review readings which as a general thing are lower than the readings first reported and as giving too much weight to past readings if the blood pressure history is taken into account.

It must be admitted that the average of all readings may not be as representative of the blood pressure of an individual as one reading under selected circumstances. Since the circumstances of the taking of an applicant's blood pressure are as a rule unknown to the medical underwriter, the problem of selecting a representative reading is difficult of solution. The use of the average of all readings has on the other hand the important advantage that most blood pressure experiences have been based thereon, and therefore reflect mortality corresponding to various ranges of "average" blood pressure. The average of all blood pressure readings can therefore be properly rated on the basis of the existing experiences. As regards the applicant, it is questionable whether the difference between the average of all blood pressure readings and the representative blood pressure is significant compared to the normal variability of blood pressure. The foregoing considerations led us to base the tentative Metropolitan ratings on the average of all readings taken at the time of application and including history within two years but disregarding obviously erroneous readings.

The use of the highest blood pressure as an index has been fully discussed by Dr. Bolt.

Dr. Bolt's "synthetic" blood pressure, or the average of the two highest systolic and the average of the two highest diastolic readings, appears to be an interesting approach to the representative blood pressure. However, in order to apply it in practice, it would be necessary to determine beforehand a large number of mortality ratios corresponding to various combinations of "synthetic" systolic and "synthetic" diastolic pressure. The ratios shown in Dr. Bolt's Table X provide only a few points for a rating schedule applicable to rating "synthetic" blood pressure.

I am not in sympathy with the implied proposal of Dr. Bolt's to rate cases which have never been 150/100 by a somewhat different method from those which have at some time shown a record of readings in excess of 150/100. The drawing of an arbitrary line of demarcation such as 150/100

leads to inconsistencies in the treatment of borderline risks such as may be illustrated by widely different ratings for a case of 149/99 and one of 151/101. It seems perhaps unnecessary to stress the fact that all mortality and other data extant indicate a gradual and continuous rise in mortality with increase in blood pressure. In practice not too many subdivisions of the rating schedule appear desirable and this consideration must of necessity lead to some inconsistencies, which should not be aggravated by the use of two different methods of rating. The use of one type of blood pressure index throughout, whether "average" blood pressure or "synthetic" blood pressure, would preclude two different methods of rating.

In the preceding paragraphs I have tried to emphasize indirectly what seem to me to be the principal requirements of the form which blood pressure ratings should take if they are to conform with the findings of recent investigators. The first is that ratings should be shown for various combinations of systolic and diastolic pressure and the second is that there should be no abrupt changes in ratings at arbitrary points. The schedule, shown on opposite page, of blood pressure ratings now under consideration for adoption by the Metropolitan is offered in illustration of these principles.

This schedule was constructed by merging the ratings derived from the Metropolitan's standard lives' experience with the ratings based on the M.I.S. findings for the substandard groups of 25-34 mm. and 35-44 mm. above average systolic. In charting the ratings for high pulse pressure we were guided by the findings of the Metropolitan experience which showed for a group with an average pulse pressure of 75 mm. a mortality of about 200% based on 33 deaths. This is corroborated by Dr. Bolt's findings on pulse pressures over 80. The ratings for low pulse pressures were based on the concensus of our Company's medical opinion which regards pulse pressures of less than 30 as abnormal.

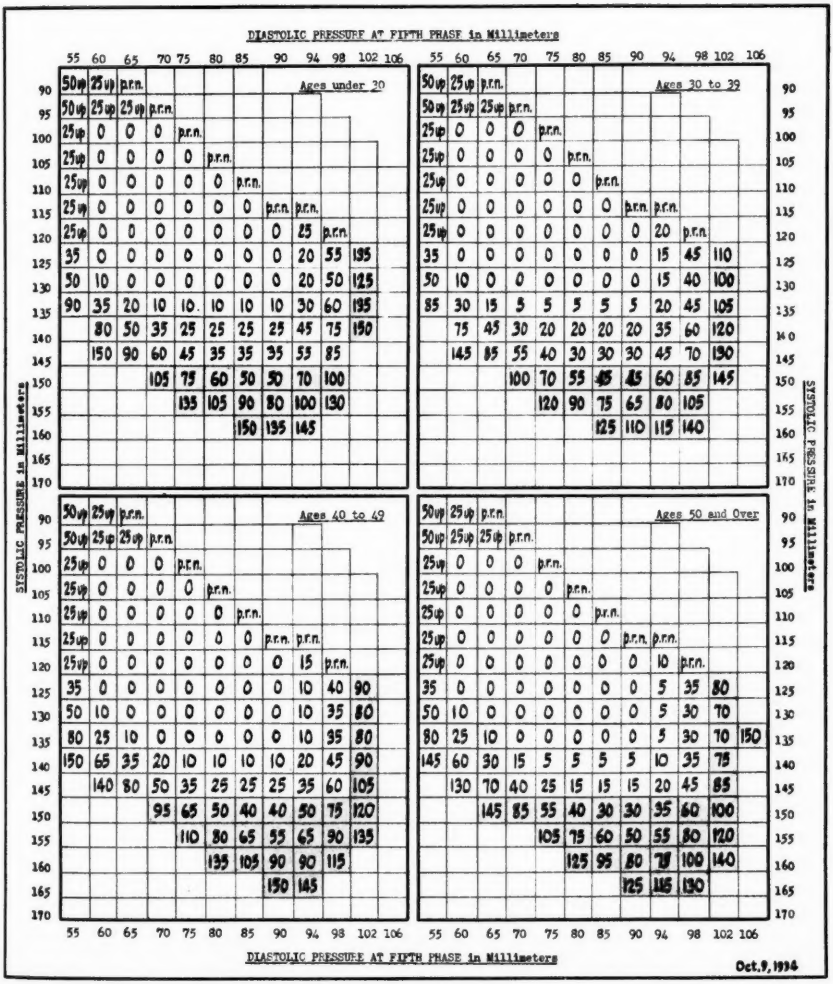
In this schedule of ratings we have given recognition to the variability of blood pressure readings by refrain-

PROPOSED BLOOD PRESSURE RATINGS

The rating should be based on the average of all present readings including history within two years, but disregarding freak readings.

The diastolic figures of the table are for the fifth phase. The fourth phase readings may be disregarded except where there is a difference of more than 10 millimeters between fourth and fifth phase, in which case the application should be referred to the Medical Division to question the accuracy of the readings. The table shows the ratings corresponding exactly to the indicated readings. **Ratings for intermediate readings should be determined by reference to the ratings shown in the table.** Readings outside of the range covered by the table call for rejection.

For ratings falling in the colored portion of the table a Home Office specimen must be ordered.



ing from rating either the systolic or the diastolic readings until about 15 mm. in excess of their respective averages. In this connection it should be noted that the true fifth phase diastolic readings are considerably lower than those so labelled by the Joint Committee in the 1925 Blood Pressure Report. The following table compares the average diastolic pressures reported as fifth phase by the Joint Committee with the Metropolitan 1925 and 1932 diastolic averages and some published fifth phase diastolic averages which there is reason to believe are true fifth phase readings.

TABLE 2

AVERAGE DIASTOLIC READINGS

Age	Joint Committee Males Ostensibly Fifth Phase 1925	Metropolitan Males Ostensibly Fifth Phase 1925	Metropolitan Males Ostensibly Fifth Phase 1932	Minn. Mutual C. N. McCloud Fifth Phase 1923	Equitable of Iowa R. G. Hunter Fifth Phase 1924
20	78.8 mm.	77.1 mm.	73.8 mm.		73 mm.
25	79.9	78.2	75.9	73 mm.	76
30	80.9	79.3	76.8	74	77
35	81.8	80.4	77.7	75	78
40	83.0	81.3	78.6	76	78
45	84.3	82.1	79.5	76	79
50	85.5	83.2	80.3	77	80
55	86.4	84.2	81.3	78	81

It appears that the 1925 Metropolitan diastolic averages contained a considerable admixture of fourth phase readings and that the 1932 Metropolitan diastolic readings, although showing considerable improvement in the taking of fifth phase readings, still include a few fourth phase readings. The same remarks would probably apply to the diastolic averages presented by other companies as ostensibly fifth phase readings, a conclusion which Dr. Bolt shares with me.

It is our intention in the near future to make a thorough study of the variability of blood pressure, with emphasis on the different causes thereof. Our tentative plan calls for a study of both the objective and the subjective causes of variability. Under the objective causes may be listed such items as faulty calibration of the instrument, the personal equation

of the observer, the tendency to record blood pressure ending with digits 0, 8 the bias which results in lower review readings, etc. Under the subjective causes would be included sex, race, build, personal habits, effects of nervousness, time of day and other factors. In this connection some recent data regarding the variation of blood pressure by sex may be of interest:

TABLE 3

AVERAGE SYSTOLIC AND DIASTOLIC READINGS BY SEX

Age	MALES		FEMALES	
	Systolic	Diastolic	Systolic	Diastolic
Under 20	116.4 mm.	72.4 mm.	112.4 mm.	71.3 mm.
20–24	120.7	75.3	116.8	73.6
25–29	121.5	76.5	117.4	74.7
30–34	122.2	77.2	118.7	75.7
35–39	123.4	78.3	121.3	77.2
40–44	124.8	79.1	123.2	77.9
45–49	126.9	80.0	127.4	80.3
50–54	128.9	80.9	130.3	81.3
55–59	131.4	82.0	133.3	81.6

The above averages are based on the Metropolitan Issues of 1931 and 1932.

It appears from the above table that the systolic averages for males are higher than those for females from 4 to 2 mm. at ages under 45, but that at ages over 45 the females' averages are from 1 to 2 mm. higher. The diastolic averages show a similar trend by sex. The conclusion seems to be that from the time of the menopause on, the average blood pressure of females is higher than that of males, which agrees with observations previously made by Dr. Riesman (6). However, until mortality statistics for blood pressure are developed separately for the two sexes, the relatively small differences in blood pressure averages by sex need not be considered in ratings.

An investigation of the different factors of blood pressure variability should lead to our concentrating on those factors which cause the relatively largest variations. It might

eventually prove advisable to so modify the instructions to the medical examiners taking blood pressure, that a control or at least a check is established on the important causes of variability.

The establishing of controls or checks on the circumstances of taking the blood pressure may enable us to distinguish temporarily elevated blood pressure from what is usually referred to as moderate "hypertension"—meaning a moderate but apparently pathological elevation in blood pressure without any other medical impairment. The following table appears to illustrate very nicely Dr. Bolt's definition of hypertention as a cardiovascular renal disease:

TABLE 4

CAUSES OF DEATH WITH INCREASING SYSTOLIC PRESSURE

Metropolitan 1922-1923 Standard Issues Traced to 1931
Percentage of Claims by Cause in Systolic Pressure Groups

Cause of Death	Systolic −4 to 4+ mm. About Average	Systolic +5 to +15 mm. Over Average	Systolic +16 to +24 mm. Over Average
Organic Heart Disease	11.4%	13.2%	15.9%
Cerebral Hemorrhage and Apoplexy	5.8	7.9	15.9
Nephritis and Bright's disease	5.7	6.6	13.9

There remains the problem of distinguishing the extra mortality associated with moderate elevation of blood pressure from the extra mortality due to the undetected inclusion of doctored cases of high blood pressure or to the undetected inclusion of other impairments. Dr. Bolt's data regarding the trend of blood pressure mortality by duration, and other data on the same point appear to shed some light on the problem. It appears from the experience presented by Dr. Bolt in Tables I, III, and VIII that the mortality of standard lives with a moderate departure from average blood pressure increases with duration. The same conclusion was reached and stated in the results of the investigation of the Metropolitan's standard lives with moderately elevated blood

pressure (5). On the other hand, Dr. Bolt's substandard experience for considerable departures from average blood pressure shows in Table IX higher mortality in the first five years of duration than subsequently. The contrary findings in the M.I.S. may perhaps be disregarded because of the probable distorting effect of the M.I.S. standard table on blood pressure mortality in the early durations. More relevant is the experience of the Northwestern Mutual (7) on lives rejected because of high blood pressure which showed high mortality in the first few years following rejection, indicating high initial mortality as characteristic of high blood pressure. This is in line with deaths from heart failure, cerebral hemorrhage and apoplexy usually associated with high blood pressure. In contrast, moderate hypertension appears to be characterized by deferred mortality, as would be expected for physiological reasons.

There are several minor points in Dr. Bolt's paper to which criticism may be directed. The comparison shown in Table X between the M.I.S. mortality ratios generally based on average of blood pressure readings with the N. Y. Life mortality ratios for "synthetic" blood pressure groups appears to be somewhat far-fetched with the possible exception of the M.I.S. group +35 to +44 mm. which does roughly correspond to the N. Y. Life "synthetic" blood pressure group 170/106—174/106. Taking into account the admitted differences between the material included in the M.I.S. blood pressure study and in the N. Y. Life's "synthetic" blood pressure groups, it seems that the conclusion as to the correspondence of the two sets of mortality ratios is founded on no better ground than similarity in figures. The conclusion drawn from Table III that the mortality in the age group 40–49 is worse than in the age group 50–59 should probably be qualified to the effect that in the latter age group the mortality refers to a smaller departure from average for that age group. The same criticism applies to Tables VIII and IX which show mortality ratios for ranges of absolute

blood pressure without emphasis on the smaller departure from average represented by the absolute blood pressure for the age group 50 and over. As regards Tables IV, V, and VII, I am afraid they would not be admitted before a court as proof of increasing mortality, though they point to this inference.

In conclusion, I want once more to congratulate Dr. Bolt on his valuable contribution to the literature on blood pressure. I also want to acknowledge the help and guidance of Doctors Christiernin and Jimenis and the great amount of work by Mr. E. A. Lew of the Metropolitan Actuarial Division in the preparation of this discussion.

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Dr. Fraser—There yet remains the discussion of Dr. Bolt's paper by Mr. Hunter. However, as the time has arrived for luncheon, I propose to carry this paper over into the afternoon and read it immediately preceding Dr. McPhedran's paper.

After luncheon there will be a showing of the Harvey Tercentenary Film made by the American College of Physicians.

It is the property of the Historical Division of Johns Hopkins University. This will be shown immediately after luncheon is over.

Afternoon Session

The Friday afternoon session opened with a showing of the Harvey Tercentenary film.

Dr. Fraser—I will read the discussion of Dr. Bolt's paper on hypertension by Mr. Arthur Hunter. (Read Mr. Hunter's paper.)

The paper by Dr. Bolt is full of interesting information and is an excellent example of cooperative effort. The enthusiasm which Dr. Bolt developed in the course of his work resulted in several of the physicians on our staff giving up many hours of their own time studying individual cases. The theme is accordingly based on the medical point of view with a 'cello obbligato by the Actuaries.

Whatever I have to say is not in the nature of criticism as I have already acted as a mentor to Dr. Bolt, but will consist principally of comments on an excellent piece of work.

His first study of the mortality based on the mean of the systolic and diastolic blood pressures again brings out the favorable mortality in cases of blood pressure below the average. Without desiring to harp upon the subject too much I wish again to point out that the average is not the normal blood pressure if the latter is taken as the point of lowest mortality. It seems that the best mortality is obtained at from 5 to 15 or 20 points below the average systolic. Should we not, therefore, in thinking of blood pressures of 15 points above the average, consider them as 25 points above the normal optimum.

In the over-average group, +5 to + 15 mm. above the "mean" blood pressure, the mortality is 116% among standard issues with a large volume of material and 174% among substandard policies with a reasonable amount of data. There is no doubt that our

medical staff were able to determine the types which should be treated as substandard by reason of a history of an impairment or of an existing minor impairment. It is clear from this and other studies that blood pressure cannot be considered alone if there is any other impairment with which it can be "hooked up", thereby materially increasing the hazard. I agree with Dr. Bolt that as the result of greater knowledge we are making a better selection, especially in taking into account other impairments which might not have been considered important a few years ago when associated with blood pressure.

One of the most significant results of the study is that a past history should not be ignored. Most companies have been too lenient with cases where there has been a high blood pressure in the past and which has been cleared up after a very careful study. The same applies to cases where there has been some impairment in the past such as a heart murmur or albuminuria, but which had apparently disappeared. I question whether this was the fault of the medical staff as much as it was the effect of competition among the companies. It has been difficult at times to withstand the pressure when another company has ignored past history after making a number of thorough tests. Unfortunately the companies could not always get at the complete history, some important facts having been concealed. There were also perfectly honest individuals whose impairment had disappeared but a recurrence was likely to take place in a material percentage of such cases.

With regard to the practice of taking the average of the systolic pressures, I have long felt that this was too liberal a procedure. When a normal blood pressure or a slightly elevated one is now found on several examinations, and there has been a record of high blood pressure in the past, it is likely that the Insured had put himself under the care of a physician after the high readings had been obtained. At the time of his new application for insurance he has probably been leading a careful life or has been "groomed" for the occasion. It seems to me that the records of high blood pressure should be given decidedly more weight than those which are average or slightly higher. It is

very difficult to lay down a rule for giving due weight to the various readings because a high blood pressure many years ago followed by a number of lower readings on different occasions over a period of years is not so significant as one in which the high readings were obtained a few months or a year ago. Such a rule would take account not only of the time elapsed since the high blood pressure but of the number of readings which had been made and the period over which they had extended.

A part of Dr. Bolt's paper in which he refers to an elevated blood pressure with a history of albumin, sugar, rapid pulse or heart murmur, draws forth a comment on the Numerical Rating System. On several occasions I have stated that the Rating System is intended to be a guide and not an absolute rule in the selection of risks. If there is one impairment, the rating represents the experience approximately of a company or of a group of companies. There are risks which are better or worse than the average and, if markedly so, allowance should be made for it. When there are two impairments the rating may be either higher or lower than the sum of the ratings for the two impairments. For example, a higher rating should be given for a heart murmur with a history of acute articular rheumatism than the total of the two individual ratings, while the reverse may be true in the case of a bartender who drank three times Anstie's limit each day. In the case of a blood pressure somewhat above the average and a comparatively recent history of albuminuria, or some other renal impairment, the sum of the two ratings might not be enough in the opinion of the Medical Director, in which event a suitable addition should be made. Such a treatment is essential under histories which might not in themselves be significant but which increase distinctly the hazard of high blood pressure alone.

Dr. Bolt—Dr. Streight asks about the average systolic and diastolic pressures used in the different age distributions in obtaining the mean blood pressure. The procedure was as follows:

The average readings of systolic and diastolic were added and divided by two. The actual formulas were $113+\times\div 3$ and $75+\times\div 5$ ($\times$=age). These mean values were then grouped in mul-

tiples of 5, which gave an average of 100 on ages of thirty and less, 105 on ages thirty-one to fifty and 110 on ages fifty-one and over. The respective average groups were therefore taken 96-104, 101-109 and 106-114. This was in accordance with the general plan outlined in detail in the book "Blood Pressure, 1925".

In Mr. Bassford's discussion he suggests that as previous blood pressure experience has been based on average blood pressure, it would be well to continue this method. We believe, on the other hand, that the mortality in certain groups with this method is such that it would be wise to approach this problem by a new method, paying more attention to the high blood pressure readings and not ignoring other impairments, past or present. Incidentally, this study indicates that material included in previous Blood Pressure studies may be analyzed more scientifically in such a way as to throw more light on the reasons for our past experience.

Mr. Bassford also suggests that a different method has been used for the borderline cases as distinct from the synthetic blood pressure method for individuals with a blood pressure 150-100 and over. Actually, this is not so. While the actuaries were working on this borderline group we were conducting experiments with the substandard material. If, however, the borderline group were to be classified according to the synthetic blood pressure method, every one of these cases would remain in the same borderline group as in none of them was the blood pressure ever so high as 150-100.

The comment is also made that the mean blood pressure method is open to the serious criticism of entirely neglecting pulse pressure. I hold no brief for the mean blood pressure method and am not myself convinced that it is necessarily an improvement over older methods. Neither am I prepared to say at present that high pulse pressure should be ignored. Nevertheless, I would like to have demonstrated to me any evidence which actually proves that pulse pressure of itself is important, where it is not associated with aortic regurgitation, elevated systolic or other definite pathology.

DR. FRASER—We now come to the last paper to be submitted at this meeting, that of the "X-ray Evidence in the Evaluation of Pulmonary Lesions in Life Insurance Medicine" by Dr. F. Maurice McPhedran, of Phipps Institute, Philadelphia. Dr. McPhedran.

DR. MCPHEDRAN—Mr. President and Members of the Association: I have great pleasure in presenting this subject. I was very glad to have it raised by Dr. Fraser and Dr. Fellows and Dr. Old of the Provident Mutual of Philadelphia, with whom I have been doing some work on these cases. The basis of our observations in tuberculosis is a group of families that has been studied in our clinic for the past eleven years. I have gone over these records carefully to confirm or to modify my impression, which is given in this paper, that after forty certain types of lesions very exceptionally showed changes and still more exceptionally were causes of disability and positive sputum.

Now, in the evaluation of these cases, the significance of tuberculosis at this age, the X-ray film has been the criterion which we have held basic. I have gone over them carefully. I have a great many typical films here and for those of you who are interested in the appearance and in the evidence in connection with retrogression and healing of these lesions, I would like to show the cases afterwards. I am going to show typical cases and I hope that those of you who are interested will ask questions if I leave any gaps, because while I am not infallible and 100% right, I am absolutely convinced, from the series of cases we have examined and followed closely with annual films, that these lesions are stable, that they are not going to increase, that they are the kind of lesion that does not affect either health or the expectancy of life.

The important thing, of course, in a problem like that is to decide what is illness from tuberculosis. Now a man may have a healed lesion and for one reason or another he may come along with a cough indicating that he may be relapsing with tuberculosis. If you allow a man to say that he has a relapse simply because of a cough and expectoration, then there is no

protection against that man being classified again as tuberculous, but if you require, in the proof of tuberculosis, either that there be present a positive sputum or X-ray evidence of progression of the lesion, then I believe that the evidence I am submitting is practically irrefutable, that is, if these men are not suffering from their tuberculosis.

Now when it comes down to putting the whole thing on the X-ray film, I should like to say at once that that film has to be adequate. There are measurements, physical measurements, that can be made of an X-ray film, and we have been working on them for some years and we have now got to the place where we can measure a film and determine whether it is within a satisfactory range of density, that it is a film that does show the densities in the chest. It is obvious that as long as X-ray films are a record of relative densities, you have to have the right density film. For example, if you are taking pictures of a steel casting, you would need one type of X-ray film; if you are taking pictures of a flower and want to show the veining in it, it is obvious you would have to use a different kind of X-ray film. The point is that for each chest it is possible to use that film that gives the maximum appearance, the maximum density change and the maximum shadow for the minimum pathological change. In other words, a film can be measured now physically in such a way as to determine whether it is a good film or bad. All that material is to be published shortly and there isn't going to be any recourse or arguments from its general trend. Therefore, when we speak of a good film, we mean a film that shows even small changes in density. That means, then, that the film is the basic criterion as to whether the patient has or has not tuberculosis.

The fundamental thing, of course, is the positive sputum. Lots of people with progressive lesions do not have positive sputums after several years' observation. We have seen that among medical students and we have seen it in those whom we have examined and have seen it in families under our supervision.

Now, then, in this paper—I don't know whether it is clear—we defined latent lesion as one that was present in the chest by

X-ray examination but wasn't causing symptoms or signs. One can see things change by watching them for a long period. We have watched cases sometimes for 4½ years and more and have seen the lesions changing and increasing without causing symptoms. Other ones advance a little and then retrogress, that is, fluctuate, without causing symptoms. That is our definition of latent tuberculosis. It is simply a lesion that may be there but is not causing symptoms.

The definition of arrested tuberculosis, that is, tuberculosis that is stable and insurable, is tuberculosis that is reduced to scar tissues that can be recognized as calcium and that do not progress over a period of years.

There are some modifying factors in classifying these cases, chiefly race and sex. I won't go into those. They are too well known to be discussed extensively.

The real question, then, from the insurance point of view seems to be, when is tuberculosis insurable, and, if so, what is the criterion? There is one way to work out that thing accurately and that is to take a group of cases and analyze them by the extent of the lesion and age at which it occurred and note the progression. Unfortunately, compiling that material is a job that would require a year's work and I have not been able to give that time. However, it can be done and I hope some day we'll get at it. I am sure it won't alter essentially the things I am saying here, but I do think it would be desirable to have that.

As to the exact measure of danger or safety that that lesion indicates, the quality and the extent of the lesion are the things that should modify one's opinion. I am just going to show some slides to show along what lines we have been working. (Showed a number of slides.)

The question may be asked, Does a patient ever break down after forty? I maintain that if you have a negative chest at forty, a chest showing the kind of scarred lesion which we have learned to regard as safe, it is very rare that they break down.

X-RAY EVIDENCE IN THE EVALUATION OF PULMONARY LESIONS IN LIFE INSURANCE MEDICINE

By MAURICE MCPHEDRAN, M. D.
Philadelphia

In prognosis, as in diagnosis, the X-ray gives basic information, because it will show changes in tuberculous lesions even in the absence of other objective evidence of instability. Moreover the extent and "quality" of the roentgenographic shadow will give considerable indication of the potentialities of the lesion. The X-ray is necessarily our chief reliance in forming an opinion as to whether a latent lesion is likely to progress to manifest illness, or whether an apparently arrested lesion may again cause symptoms and physical signs; in ascertaining, that is, that a pulmonary lesion has attained a dependable stability.

Certain modifying factors should also be taken into consideration with the X-ray evidence. Of these the most significant are age, race, sex, personal history and family history. For example, a young man in the early thirties with a strand-like apical lesion may be a good risk; another young man with a similar lesion but with an immediate family history showing a high rate of morbidity and mortality from tuberculosis may be a poor risk. Again, statistics show that tuberculosis occurs somewhat more frequently and more destructively in women than in men during adolescence and early adult life, while at middle age the death rate is higher in men than in women. Colored adults are considerably more prone than white adults to suffer rapid spread with acute illness and high mortality. In them also childhood-type infiltrations may cause a highly fatal disease. These lesions in the white race are usually seen before puberty and except in infancy are commonly

benign; among white adults our chief concern is with tuberculous lesions that arise in and spread downward from the apex.

Serial studies of families in which tuberculosis has developed are the basis for this paper. These studies have borne out and amplified pathological observations[1] and clinical experience showing that many persons with tuberculous lesions may live to die of other causes, often at an advanced age. Many pulmonary lesions found at autopsy, including extensive apical infiltrations, never seriously disturbed health, and in many instances were not discovered during life. Among groups who had undergone treatment for tuberculosis, Heise and Brown[2] found certain percentages of former patients alive twenty and forty years after dismissal from the sanatorium. It is noteworthy that figures based on experience before the use of the X-ray are more unfavorable than those based on groups that have had the benefit of roentgenographic observation in securing earlier diagnosis, in supervision carried to more effective healing, and in accurate follow-up of patients who have returned to work and continue to live under normal conditions without suffering limitations from their former disease. Our present problem is to evaluate the probable potentialities of lesions, both those that have apparently been arrested by treatment, and those latent lesions that, having never caused noticeable symptoms or physical signs, are discovered incidentally to a routine examination.

The prognostic significance of the size of apical lesions varies with age. In a group of people of various ages with latent apical lesions, many of them observed for from six to eight years[3], we found that after forty most latent lesions were stationary, although in approximately 6 per cent. previously latent lesions caused symptoms and physical signs after forty. In younger persons with small lesions extending below the second posterior rib, clinically manifest tuberculosis developed in 6 per cent.; it developed in 25 per cent. of those with latent lesions occupying over

half or more of the apex above the level of the clavicle, and in approximately one-half of those with latent lesions extending below the clavicle. We believe it may be assumed that persons less than forty years of age who have latent apical lesions approximately equal in size to one-half of the apex above the clavicle are actually in need of treatment appropriate to clinically manifest tuberculosis unless the lesion has the anatomical characteristics of stability confirmed by repeated X-ray examinations. Further analysis of the conditions under which spread occurs, especially in older age groups, would be desirable.

In adults[4] an apical lesion may be first seen as mottling, which may be sharply defined or ill defined and more or less confluent. It may appear as strands or salients, sometimes sharply delimited from the surrounding lung field, sometimes "fuzzy" in outline. Or it may be seen as a homogeneous consolidation involving the whole apex or a small part of it, or occupying the third and fourth axillary spaces. The less sharp the outline of the density, the more it fades imperceptibly into normal lung tissue, the greater will be its capacity for change, whether advance or retreat. Sharply demarcated strands, wholly limited to the area above the clavicle are rarely significant in a man in middle life. In a young man, on the other hand, especially in the second and third decades, they are evidence of a severe infection that may spread without warning, sometimes as a result of apparently slight strain.

In a young adult no apical lesion should be considered a scar unless it consists exclusively of spots of calcium and sharply defined dense strands. The more recent and more prolonged the contact with sputum-positive tuberculosis, the nearer the patient to the early twenties, and the larger and less sharply delimited the densities, the more uncertain the prognosis. Lesions of this poorly demarcated type, even if they remain unchanged over years, cannot be regarded as stable. Severe apical lesions often appear as wedges or irregular masses resulting from the confluence of the spots

that composed it; such lesions are likely to spread rapidly with cavity formation. A fairly dense cloudy infiltration with a flocculent or definitely "fuzzy" periphery is always dangerous. Such lesions respond well to prompt and adequate treatment, but they should not be regarded as dependably scarred until they appear as sharp strands that remain unchanged for weeks under close radiographic observation. Only then can treatment be safely relaxed, and the former patient considered a "safe risk" from any point of view on his return to the activities and strains of normal life.

Whenever the densities composing a lesion are close-set the most important objective of X-ray examination is to exclude the presence of small rarefactions due to cavity. On the one hand these are often obscured, and on the other hand, they constitute a most serious threat. The presence of any verifiable cavity, however small, makes the lesion as a whole prognostically uncertain. No doubt occasional cases with verified cavities would be acceptable risks on the basis of serial films showing a stable condition over years. Each case of this sort should be given separate careful consideration. *Exposures of various densities, and sometimes views from more than one angle may be necessary to rule out excavation. They may also be required to exclude the possibility that overlapping strands are mistaken for cavity walls. In the latter event, serious injustice may result from inadequate or inaccurate X-ray examination.* For example, the protection of life insurance, or a school teacher's position, often depends upon exact exploration of the components of a lesion. These considerations of X-ray technique and interpretation are equally important whether a lesion that has not been the cause of symptoms is discovered incidentally, or whether the present and future stability of a formerly clinical lesion is in question.

When a lesion has given rise to symptoms in the past it is desirable to review all X-ray films showing its maximum density and its subsequent course. The sites of former cavities should be carefully scrutinized. In some cases the walls of a cavity become almost imperceptible long before it starts

to contract, and it is often possible to follow in serial films the contraction of such cavities from a diameter of several centimeters to two or three millimeters. At this point a stellate scar often replaces the cavity. When the center of this is sharp and dense, and the radiating strands sharp, with no suggestion of fuzziness, a dependable scar has usually been formed. The most exact adaptation of quality of film, position of the patient and level of the tube is essential in resolving such problems.

Alone or in association with the apical lesion there may be calcified pulmonary nodules[5]. These are of no significance at any age; only an unencapsulated caseous focus in an infant is serious. Calcified tracheobronchial lymph nodes in young adults are indication of severe exposure to tuberculosis in childhood, but they are in themselves of no signifiance if all possibility is ruled out that contact with open tuberculosis is not being continued or is not repeated. Calcified nodes are often vary large without any history of illness ascribable to tuberculosis. They appear as granular, stippled or dense shadows in the hilum shadow or, as shown by oblique films, in the mediastinum, or in both; the pulmonary nodule may be obscured by dense structures. If calcified nodes appear in association with an apical lesion, exposure to contagion has presumably occurred again, or has been carried over into adolescence through contact with the original source of contagion or another. Care should be taken to ascertain that the apical lesion is stable and that contact with tuberculosis has been completely broken and will not be renewed. In such cases the family and personal history, and the age of the person examined, must be carefully weighed.

When calcified lymph nodes are seen alone the most important point is to consider the most recent probable date of exposure in relation to the subject's age. The closer the age to adolescence or the early twenties, the more likely that contact may still be continued with the original source of contagion or with a new one developing within the same

family group. There is no evidence, however, that such persons are particularly liable to develop tuberculosis once exposure has definitely ceased.

When X-ray films show no pathological shadows, with the possible exception of calcified pulmonary nodules or calcified lymph nodes, inquiry should still be directed to ascertain that there has been no recent exposure to tuberculosis. This is probably the more urgent in young adults, who may have been exposed during adolescence. Much of the morbidity and mortality from tuberculosis is caused by the development of latent lesions acquired under 30 years of age.

When a basal lesion is associated with an apical lesion in the same lung, it is important to recognize the non-tuberculous origin of the former and of any disability it may cause. These lesions are often associated with stable lesions in the apex, particularly in middle age. There is no evidence that exacerbations of the basal lesion will activate or aggravate a tuberculosis lesion in the same lung. Many patients with arrested apical lesions, but with cough and expectoration caused by a non-tuberculous basal lesion, have been erroneously described as suffering from the benign tuberculosis of middle age. The exact prognostic significance of basal lesions is as yet unknown. These lesions, both as they are associated with tuberculous lesions and as in themselves a cause of ill health, deserve more study than they have hitherto been given.

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DR. FRASER—The discussion of this paper will now be undertaken by Dr. Herbert Old, Medical Director of the Provident Mutual Life Insurance Company of Philadelphia.

DR. OLD—(Read paper.)

When I was in Westminster Abbey I was struck not only with the fact of how many of the memorials had been erected in honor of great leaders in the British army and navy, but also with the fact of how many of these leaders had won their distinction in wars against the French.

I came away with the impression that the British really owed the French a debt of gratitude for the opportunity offered them by the French to achieve greatness.

If the profession of medicine had a similar shrine for its heroes, it would be interesting to find how many had won greatness in the constant warfare between the human race and the tubercle bacillus. One might almost say that the medical profession, and the many various organizations that have been carrying on this combat, are indebted to this bacillus for the opportunity it has afforded them to achieve great distinction.

The No-man's Land, or undiscovered territory, is the WHERE, HOW and WHEN this bacillus can be controlled in the same manner we can prevent yellow fever, malaria fever, typhoid fever, etc. We practically know that all living infants are born without the taint, that about ten per cent. during the first year, fifty per cent. by the sixth year and over ninety per cent. by the eighteenth year, harbor the bacilli in their bodies. However, only ten per cent. are seriously infected by the disease. When we can explain why the one individual has the disease and the nine escape, we will take a great step forward in winning the conflict.

Tuberculosis is a disease which appears at any time of life, from infancy to old age. It assumes various subtle and complex guises. When the cause for a diseased condition is indefinite or unknown, it is well to think of tuberculosis.

Quite a number of trenches have been dug within which one can obtain a firing stand and shoot effectively at the enemy. We know:

1. The bacillus which causes the disease and how to detect it.
2. Something about the necessary balance between allergy and immunity and its bearing upon arrest or progress in the lesion.
3. That there is a great difference in the prognosis of the juvenile and adult types of lesions among the white race.
4. That it is now possible to differentiate between inactive or benign and active or progressive apical lesions.
5. That sub-apical lesions are of greater danger not only to the individual himself but to others in his acting as a carrier.
6. That the soft pedal has properly been placed on hereditary influences, both as to seed and soil; and that more and more emphasis is being placed on re-infection and super-infection by coming in close contact with an open case of tuberculosis or a positive-sputum carrier.
7. That the children in families where one or more cases of pulmonary tuberculosis have occurred stand double the chance of being seriously infected, accompanied by a high mortality, than those in families with no such history.
8. That where one consort is affected, the other is much more apt to contract the disease than when neither is affected.
9. That the analytical statistics of the life insurance companies, covering a large number of cases over a long period of time, have shown the potential dangers attached to underweight, family history, rapid pulse, personal history of pleurisy, or hemoptysis, or fistula in ano, and frequent attacks of upper or lower respiratory infections, especially in those under age forty. In fact, judging by our disability claims, it looks as if this age should be extended to forty-five, inclusive.

PROVIDENT MUTUAL LIFE INSURANCE COMPANY OF PHILADELPHIA

Disability claims on cases for pulmonary tuberculosis—Period 1922 to 1933, inclusive.

% by Number	% by Amounts
15+%	12%

BY AGE OF OCCURRENCE

Under 21	21–30	31–40	41–50	51–60	60+
1	120	89	28	5	1
or	or	or	or	or	or
−½%	49+%	36+%	11+%	+2%	−½%

For Disability regarding underweight—

1. We required 11 lb. more than minimum weight for standard insurance between ages 16–29, inclusive, and 16 lb. more in applicants aged 30 and over.
2. Irrespective of favorable weight, we issued no Disability in applicants under age 30 with one case of tuberculosis in family history, and those under age 45 with 2 cases in family history.

10. That the most effective prevention consists of early diagnosis in order to prevent cases not only from falling into the category of "damaged goods," but from being carriers of the infection to others.

In the past, the generally accepted criteria for the diagnosis of pulmonary tuberculosis were the five formulated by Dr. Lawrason Brown, of the Trudeau Sanatorium. His suggestion that at least two of these be present appears to have been well founded, judging by the large number of wrongly diagnosed patients sent to the clinics and sanatoria for tuberculosis.

In the consideration of this paper and demonstration given here on X-ray evidence in the evaluation of pulmonary lesions in life insurance medicine by Doctor McPhedran we are concerned with only two of the criteria, namely,

1. Localized râles above the third rib in front and above the spine of the scapula behind (brought out by expiratory cough).
2. Parenchymatous X-ray densities above the third rib.

Physical diagnosis in lung conditions has always been and continues to be, judging by our claims for disability, the most

difficult of medical arts to the average practitioner of medicine. We now know, thanks to the radiologist, that the early lesion in adult tuberculosis begins below the clavicle and spreads up to the apex, and does not start at the apex and spread downward. By waiting for the râles to be elicited at the apices, one would have to delay the diagnosis until a frank open lesion or possibly a cavity was present.

In fact, radiology has now become, together with the tuberculin test, the main method of making a diagnosis of exposure to pulmonary tuberculosis in the schools and colleges and in the hospitals for the internes and nurses. The present slogan seems to be: "Tuberculosis is seen, not heard." This, together with the slogan adopted by the Anti-Tuberculosis League, "That every case of the adult type of lesion means contact with an open and sputum-positive case," is going to be the main factor in continuing to lower the mortality from this disease.

I know that each of you will consider, as I do, that this presentation and demonstration by Doctor McPhedran is truly a masterpiece and that he has proven himself to be not merely a rifleman but a sharpshooter, as evidenced by the large number of bull's eyes he has made in his ascertainments of the roamings and ravages of the tubercle bacillus. This paper should serve as a reference manual to all Medical Directors in their interpretation of the X-ray plates submitted to them for consideration.

To attempt to add to or subtract from the etching he has so vividly drawn would be like attempting to paint the lily. It can not be done.

I feel sure you were struck with the emphasis he placed upon age, family history, past personal history and clinical signs in the evaluation of the X-ray findings, and in this he proves himself to be a man of science with an unusually broad outlook.

We should, of course, keep in mind that even though we may feel satisfied that the lesion has been arrested as regards the occurrence of toxemia or anaphylaxis, we have present in the lungs a scar, atelectasis, fibrosis and emphysema. I would say that if we wished to express in fractional form the true evalua-

tion of the seriousness of the pulmonary lesions resulting from the various infections, especially found in those forty years of age or over, the numerator might be arrested and inactive tuberculosis, or pneumoconiosis, or bronchial asthma, or fibrosis following frequent upper respiratory infections, or bronchiectasis. The common denominator would be the amount of emphysema present plus the effect on the right side of the heart. For, as increased intra-pleural pressure becomes more pronounced, a rise in venous pressure will tend to cause hypertrophy of the right ventricle, and eventually progressive heart failure.

In the electrocardiogram, such an effect on the pulmonary circulation is shown by right axis deviation and changes in the T wave in Lead III or Leads II and III.

I felicitate the Committee on having this important subject presented so forcibly and clearly before the Association, and personally feel most grateful to Doctor Fraser for the privilege of opening the discussion.

DR. FRASER—The discussion will be continued by Dr. Haynes H. Fellows of the Metropolitan Life Insurance Company. Dr. Fellows.

DR. FELLOWS—Many life insurance companies have been reluctant to accept any applicant who shows any evidence of a tuberculous lesion, even though healed. Due to the accumulated evidence gained by use of X-rays in the past ten years, we believe that with wise selection it may be possible to select the best risks in this group. The reason for this optimistic expression is that we know now that some persons who have had pulmonary tuberculosis live to a ripe old age.

Proof:

1. Many persons, age 65 and over, by X-ray examination show scars of an old healed pulmonary tuberculosis; some know that they have had the disease, some do not.
2. Post-mortem examinations on elderly persons dying of

diseases other than pulmonary tuberculosis often show the presence of healed pulmonary tuberculosis lesions.

Both groups 1 and 2 from the standpoint of pulmonary tuberculosis alone would have been insurable risks at some time previous.

Importance of X-ray Examination:

Whenever pulmonary tuberculosis is suspected by history or physical examination, our most accurate single method of determining its absence or presence is by an X-ray of the chest.

Age:

If the applicant is 30 years of age or over and the X-ray is negative, the probability of future development of tuberculosis is very slight. Repeated statements have been made by various authorities on this subject; that if a person reaches 22 or 23 years of age without having pulmonary tuberculosis, he or she almost surely will not contract the disease. From our experience in watching the development of pulmonary tuberculosis in Home Office employees, we would place the age a little higher, at about 30, beyond which the chance of developing pulmonary tuberculosis is very improbable.

Family History:

The significance of the presence of tuberculosis in a family is the increased opportunity of exposure and infection of other members of the family. Therefore, a positive family history should require greater care in the selection of applicants less than 30 years of age, particularly those with continued exposure. If the X-ray of a contact is negative, and the individual is over age 30, logically there is no sound reason for rating the applicant.

Healed Primary Complex or Evidence of a Healed First Infection:

Since 1926 we have taken serial X-rays on a group of 3,000 Home Office employees as part of a study on the develop-

ment of adult pulmonary tuberculosis and we have seen several cases develop this disease. Our experience leads us to believe that the presence of a healed primary complex is of no significance as far as the development of adult pulmonary tuberculosis is concerned.

Therefore, when the X-ray film of the chest shows—

(a) No tuberculosis at age 30, the case is safe.

(b) Healed primary complex, the case is as safe as a normal chest.

Main Problem:

How separate the insurable applicants who have had pulmonary tuberculosis from the uninsurable?

At present state of our knowledge an X-ray examination is essential.

If X-ray is negative, we believe it is safe to eliminate the presence of a tuberculous lesion in practically all cases. If it is known to be a fact that the applicant has had pulmonary tuberculosis in the past and the present X-ray is negative, the proper procedure will be given shortly.

If X-ray examination reveals a tuberculous lesion, usually only the minimal lesion should be considered for insurance.

Definition of Minimal Tuberculosis by X-ray:

1. Slight involvement without cavity.
2. Small part of one or both lungs, "Total volume of involvement regardless of distribution not to exceed volume of lung tissue lying above the second chondrosternal juncture and spine of 4th or body of 5th dorsal vertebra."

Moderately Advanced:

Available figures show that moderately advanced cases have a much higher mortality than the minimal cases. Possibly some persons with moderately advanced lesions can be accepted now with appropriate safeguards.

If X-rays are positive for tuberculosis, there are several factors to consider.

Sex: Females—The peak mortality among females who contract tuberculosis is reached at age 25, following which there is a marked drop which is sustained until about age 55, when there is a second rise.

Males—Mortality increases steadily and peaks at about age 55 to 60.

Therefore, mortality is worse among females 25 years or less than among males of this age. Past 25 there is no reason for anticipating as high a tuberculosis mortality among females as for males and certainly no logical reason for an excessive rating for females simply because they are females.

Proof: Mortality curve U. S. Reg. Area and Ind. Policy-holders.

General Condition:

Those applicants who have had tuberculosis should have a satisfactory general condition with average or better weight at the time of application.

Proof: Weight and History of Tuberculosis—Medical Impairment Study, 1929. The mortality decreased as the weight (average and over) increased, and the mortality in those with Sanatorium treatment or knowledge of having had tuberculosis in the average and overweight group was satisfactory.

History of Sanatorium or Other Medical Treatment for Pulmonary Tuberculosis:

Some applicants whose X-rays are positive for pulmonary tuberculosis have taken treatment at a Sanatorium and others have not. Among discharged sanatorium patients highest mortality occurs (a) in the first five years, (b) in advanced cases, and (c) in minimal lesions with positive sputum.

Proof: 1. Post Sanatorium Histories of Tuberculosis Cases, Whitney and Myers—Amer. Rev. of Tuberculosis, V. 21, p. 691.

2. Certain Factors Influencing the Mortality of Sanatorium Treated Cases of Pulmonary Tuberculosis, Langmuir, Williams, and Pope—New England Jo. of Med., 7-5-34, p. 26.

Abstract of Above:

Studies made on after-histories indicate that the experience is quite unfavorable from the insurance standpoint for applicants who had tuberculosis in an advanced stage or who had minimal tuberculosis with a positive sputum. The mortality in the first five or seven years after discharge from the Sanatorium is rather high. Women have higher survival rate than men. While it may be difficult to tell from an X-ray when a lesion becomes inactive and healed, we can allow the most unfavorable cases to be withdrawn from the treated cases by waiting five years after treatment has ceased, at which time the case may, of course, be considered to be healed and inactive.

Those Without Knowledge of Having Had Pulmonary Tuberculosis:

Many persons who, at post-mortem or X-ray examination, show evidence of having had pulmonary tuberculosis, never knew that they had had this disease. We have at the Home Office many cases showing all stages of the disease discovered accidentally who do not remember ever having been ill. They have continued at work under careful observation, many are in late adult life and so far there have been no deaths from tuberculosis among this group.

Therefore, among those who have cured, the longer the period since cure the better, with five years minimum, and among those who have not cured, satisfactory X-ray and general condition should be required.

Consideration of X-ray Findings:

Satisfactory Findings:

1. Healed primary complex considered the same as normal adult chest, i. e., no rating.
2. Stringy, fibrous or calcified lesions safest because they signify healing.

Rejected or postponed:

1. Soft, fluffy, flocculent, homogenous or other lesions (cavities) seen for the first time.

Rating: Pulmonary Tuberculosis—With No Physical Signs or With Only Slight Deviations From the Normal.

	Present X-ray Satisfactory		No Present X-ray	
	Minimal	Mod. Adv.	Statement* from Sanatorium (Minimal Tb.)	No Statement
Age 30–39				
Less than 5 years..	D	D	D	D
5–7 years	75	D	100	D
7–10 years	35	D	75	125
10–15 years	15	D	35	75
15 years or more....	15	D	15	35
Age 40 or over				
Less than 5 years..	D	D	D	D
5–7 years	30	D	75	D
7–10 years	15	D	35	75
10–15 years	10	100	15	35
15 years or more....	10	50	10	15
With no history of tuberculosis				
Age 30–39	35	D		
Age 40 and over....	15	100		

*If statement shows Moderately Advanced Tuberculosis, get X-ray or decline.

Dr. Fraser—Dr. McPhedran, do you care to close the discussion?

DR. MCPHEDRAN—Of course, every time one touches on any one point in this matter, it opens up endless avenues of discussion. The only thing one can do with it is to try to piece it out. No one topic of this can be closed completely.

I think we are all agreed on the main outline of this problem. There is just one point that Dr. Fellows brought up. Dr. Fellows means he won't insure them at present unless these lesions had healed to a minimum. Isn't that true, Dr. Fellows? That is the only point I was not clear on. So many cases of extensive and dangerous-looking lesions at 30 will come through at 35 and 40 with sharply healed lesions that do not change.

In connection with the other point that Dr. Fellows raised, I am thoroughly in agreement with him that a patient at age 30 with negative film makes a reasonably good risk, no matter what the family history is.

Dr. Old raised the question of classification and the importance of some of his criteria of tuberculosis. Of course, rales can persist in a chest in a man who has absolutely no symptoms and is not sick with the lesion for years.

Now there is just one other point in these serial follow-ups. Dr. Old quoted the old statistics and Dr. Fellows raised the question about the X-ray and how it had changed our evaluation of tuberculosis from the insurance point of view. The point is that we haven't got large figures on this, but we have got 100% evidence on a good many of them. I have followed some of them over a period of years and some of those living at age 40 have gone through to 60 and up and died of other causes. There is a lot of post-mortem and other evidence that these people can go through and die of other causes.

There is only one other point that bears on that statistical showing. If material could be analyzed on an extent and age period basis, I think it would give important evidence, and that would give a standard that would be very valuable. If that were taken up, I think you would be getting something more useful than any other thing.

A NEW ALIMENTARY DEXTROSE TOLERANCE TEST

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(Publication authorized by Committee on Publication—Dr. Robert M. Daley, Chairman)

In a paper presented this Association at its 1931 (Hartford) meeting we included some experimental data obtained in the process of exploring the possibilities of improving the alimentary dextrose tolerance test for the purposes of life insurance selection. Since then we have continued experimentation, and in this paper propose to summarize our results in the form of a new test which seems to be more specific and reliable than the older tests for differentiating between diabetic and non-diabetic conditions.

In the previous paper we quoted contemporary authorities to connote the diagnostic limitations of sugar tolerance tests, discussed the underlying physiology and gave directions for a test with 75 grams of glucose over a two-hour period. The present paper is, therefore, confined to a brief discussion of the differences in physiology between the older and the new tests, to describing the new test, and to illustrating its criteria for distinguishing between the more usual types of glycosuria.

Any discussion of the older alimentary dextrose tolerance tests must be based on the consideration that each test is a physiological experiment which at some definite time measures the difference between the rate at which ingested sugar enters the blood and the rate at which the body either oxidizes, stores, or excretes it. As measurements of this kind are inevitably affected or vitiated by more or less uncontrollable complications like diuresis, hydration, fatigue, psyche, digestion, etc., we have concentrated our attention on the various factors in the practical application of sugar tolerance tests which are known to influence the accuracy of results. From the work of previous investigators and our own

experience it is apparent that the chief factors are: (1) The time it takes to make the test, (2) the antecedent diet or food habits of the subject, and (3) the rate of absorption from the intestine of the ingested glucose.

The Time Element

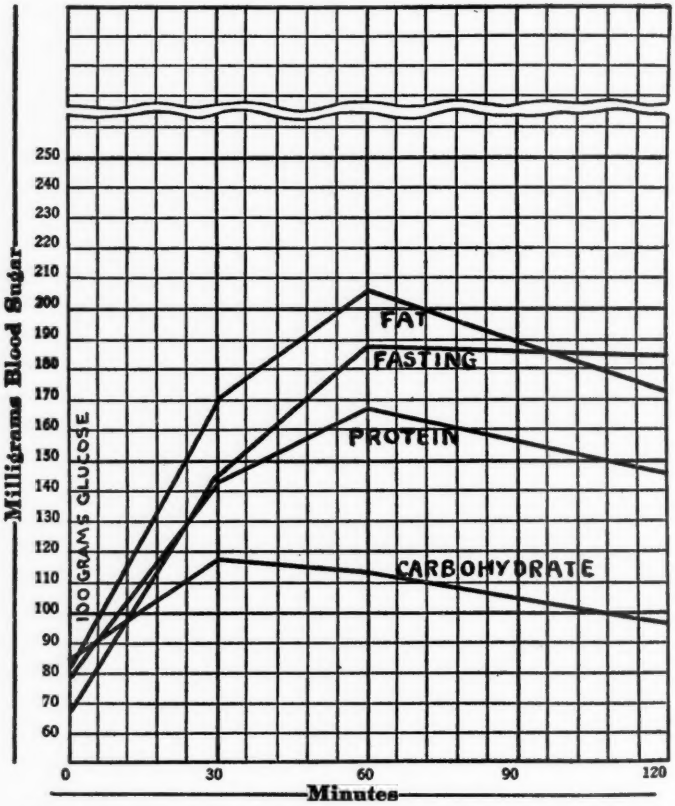
As usually performed, it takes two or more hours to make a sugar tolerance test, although it is clear that there are very definite advantages in making the period of physiological experiments of this kind as brief as possible in order to exclude certain external accidental or secondary effects such as psychic complications like anxiety and impatience, and physical discomforts like hunger, etc., which may affect the results. In our previous paper[1] we gave the details of a procedure for testing sugar tolerance with a 75-gram dose of glucose which lends itself to limiting the period of the test to two hours when a return of the blood sugar to within 20% of the fasting value is counted as the criterion for normals. In a very considerable amount of data accumulated with this test no case gave any more definite information about the sugar tolerance of an individual at the end of three or four hours than at the end of two hours. In fact, the results seemed less obscure at the end of the shorter period in some of the cases. In every instance the patients preferred the shorter test, and the advantages of abbreviating the test period have become still more apparent with the new procedure, which takes only an hour to complete. Viewed from the practical standpoint of the expediencies of life insurance practise the shorter period of the new test has, furthermore, the special advantages of not only being much more convenient for applicant and examiner but also of reducing to a minimum the possible effects of any attempt an applicant may make to interfere with the honest result of a sugar tolerance test.

The Effects of Prior Food

Another and more important influence on the results of the older sugar tolerance tests is the antecedent diet or food habit of the subject. A number of authors have called attention to

this factor, but none so vividly as Sweeney[2] in this country, some of whose data are shown in Chart 1. These curves show the average responses to dextrose tolerance tests of groups of healthy young adults living on exclusive diets for 48 antecedent hours.

CHART 1



Data of Sweeney showing effects of 48 hour exclusive antecedent diets on Dextrose Tolerance Tests. Averages of 4 normal young adults on protein, 5 on fat, 8 on carbohydrates and 5 on nothing but water. Only those on carbohydrates gave a normal response.

The curves are particularly suggestive when it is remembered that all of the subjects gave normal curves when tests were made after antecedent mixed diets and that reversals of the curves occurred with the same subjects on changed diets. It is also to be observed that although all of the subjects were known normals, those who took nothing but water and those who ate exclusively fats or proteins gave misleading results, while only those normals who ate carbohydrates gave normal responses to the tests.

Sweeney postulates that the differences in his results are due to variations in the stimulation of insulin by the different diets and concludes: "The dextrose tolerance test as usually employed has been shown to be materially influenced by different antecedent foods or diets. * * * Some of the curves believed to be peculiar to different pathological conditions may result from the diets."

In opposition to Sweeney's results with the older test, the theoretical basis of the new test implies that its results are not influenced by antecedent diets or food habits. We therefore tested a number of subjects under the conditions of their accustomed food habits in accordance with Nielsen's[3] conclusion that there should be no alteration of a subject's food habits prior to dextrose tolerance tests. It was thought that the information secured in this way would conform with the general run of actual conditions in clinical and insurance practise better than the information obtained under the less usual conditions of depriving subjects of their accustomed food and restricting them to unaccustomed exclusive diets.

Two groups were made of sixty-five males and females, running from 18 to 64 years. Of these, some were known to be living on more or less exclusive diets. The first group consisted of six diabetics and forty-five non-diabetics. All of the members of this group were tested, not after the usual overnight fast, but at random times and regardless of what or when they ate. The second group consisted of fifteen diabetics, all of whom were tested, not at random times, but only after an overnight fast. In both groups all of the new tests gave perfectly typical results regardless of food conditions. Hence, unlike the older,

the results of the new test are not affected by antecedent diets or food habits.

Under the special conditions of life insurance practise it is not always expedient or practicable to carry out sugar tolerance tests after an overnight fast. The independence of the new test with respect to antecedent diets, therefore, offers much greater convenience and accuracy. For the sake of uniformity sugar tolerance tests should always be carried out after an overnight, or equally long, fast, whenever practicable. Whenever impracticable, the new test is undoubtedly safer and more conclusive than the older test because it is the course of the blood sugar after a second dose of glucose that distinguishes between diabetic and non-diabetic conditions, and it appears that this criterion of the new test does not tend to change with variations in the time or nature of food taken prior to the test.

The Effects of Absorption

Of all of the factors which influence the results of sugar tolerance tests, the absorption of the ingested glucose from the intestine is by far the most troublesome. As long ago as 1915 Woodyatt, Sansum and Wilder[4] held that the irregularities inherent in absorption of the ingested glucose from the intestine vitiated the results of alimentary sugar tolerance tests and they therefore proposed an intravenous procedure designed to avoid such irregularities.

Following their work, Beeler, Bryan, Cathcart, and Fitz,[5] after reviewing earlier experimental work, proposed "An Improved Alimentary Glucose Tolerance Test" in which the usual procedure was modified by emptying the stomach with a tube an hour after the ingestion of glucose. By measuring the difference between the ingested glucose and the amount thus recovered from the stomach they aimed to ascertain the amount of glucose actually absorbed, and interpret their results on that basis. They summarize their interesting experiments, which cover some of the unavoidable complications of absorption, as follows: "Clinical observations emphasize certain obvious defects in the alimentary glucose tolerance test. * * * They have not received sufficient

attention. The absorption of glucose from the intestinal tract is a complicated process. The rate of absorption varies with each individual within wide limits. * * * Glucose, in between 10 and 20% solution, disappeared most rapidly from the stomach."

Their conclusions are of special interest when related to the concentrations of glucose employed in prevailing sugar tolerance tests which, according to the information elicited by our 1931 questionnaire, vary more on this than they do on any other point, although the point is often obscured by insistence on other details, like the time allowed for drinking the glucose solution and its palatability. It is typical of present practise to give 100 grams of glucose in about 250 cc. of water. Some try to avoid the hypertonic effects of this 40% solution by giving less glucose and others by giving more water. On this point a recent study of Magers[6] confirms the findings of Fitz and his co-workers because no consistent differences appeared in the blood sugar curves of twelve subjects who were given 50 grams of glucose in 15% solution, orally and intraduodenally. In line with the experimental evidence that the accuracy of sugar tolerance tests is least likely to be influenced by irregularities of absorption when concentrations of about 15% are employed, the new test employs two 50-gram doses of glucose in 15% solution.

The Paradoxical Law of Dextrose

On the basis of animal experiments Allen[7] enunciated his paradoxical law as follows: "The paradoxical law of dextrose distinguishes sharply between diabetic and every type of non-diabetic animals. * * * Limits of tolerance in diabetic animals are real, not apparent. Just the opposite of the paradoxical law. * * * Whereas in normal individuals the more sugar given the more is utilized, the reverse is true in diabetes."

Allen was the first to suggest that the law might have diagnostic value, and Hamman and Hirschman,[8] in the fourth of their pioneer studies of blood sugar deal with "The effect on the blood sugar of the repeated ingestion of glucose." In this study they include protocols which show the effects on the blood sugar of diabetics and non-diabetics when a similar dose of glu-

cose is ingested three hours after a first dose. This effect has also been studied abroad under the name of the Staub[9]-Traugott[10] phenomenon and other names, and the trend of these studies is illustrated in the recent text of Grafe[11] by blood sugar curves showing the effects of repeating a 20-gram dose of glucose one and a half hours after the original dose.

In an effort to differentiate between the glycosurias of diabetes and hyperthyroidism Rabinowitch[12] made a particularly interesting application of Allen's paradoxical law by giving diets of constant composition with respect to protein and fat, but increasing the amounts of glucose. According to his protocols 10 grams of glucose were given every hour for ten hours one day, 20 grams similarly the next day, and 30 grams on ten succeeding days.

It thus appears that under varied experimental and clinical conditions many observers have independently established the fact that normal human beings react to repeated doses of glucose with less, or little or no change of glycemia but that diabetics react with distinct hyper-glycemia. The underlying physiology has been satisfactorily explained by MacLean and de Wesselow,[13] and very well stated by Foster[14] in summarizing his own experiments: "We interpret this fact as meaning that the first dose of glucose stimulates the insulin-glycogen mechanism to such activity that the normal organism is then able to deal with any amount of glucose without becoming hyperglycemic." To this it may be added that diabetics react with distinct hyperglycemia because the insulin-glycogen mechanism fails.

We have, therefore, tried out the possibilities of the paradoxical law by varying the experimental conditions under which repeated tests were made on the same subjects, and have elsewhere presented a summary of these tests. They gave no evidence of being in any way affected by antecedent diets or irregularities of absorption, and very definitely indicated the practicability of adapting the paradoxical law to routine clinical use. They also enabled us to define optimum conditions, and formulate a simple and convenient procedure which has since been applied to more than four hundred individuals under usual clinical conditions.

The One-Hour Two-Dose Test

Procedure

We perform the test by dissolving 100 grams of glucose in about 650 cc. of water. This solution is then flavored with lemon and divided into two equal doses containing 50 grams of glucose in about a 15% solution, which are served cold and are easy to drink within a minute. We also keep at hand three containers with our preservative[15, 16] against glucolysis for the blood specimens and three containers for the urine specimens, making it a point when collecting these to have subjects empty the bladder as completely as possible. The following steps are taken after a fast, preferably overnight:

A

Collect (A) fasting blood and urine samples and give the first dose of glucose, allowing one to two minutes for its ingestion.

B

Thirty minutes after ingestion of the glucose, collect (B) blood sample and give the second dose of glucose, allowing one to two minutes for its ingestion.

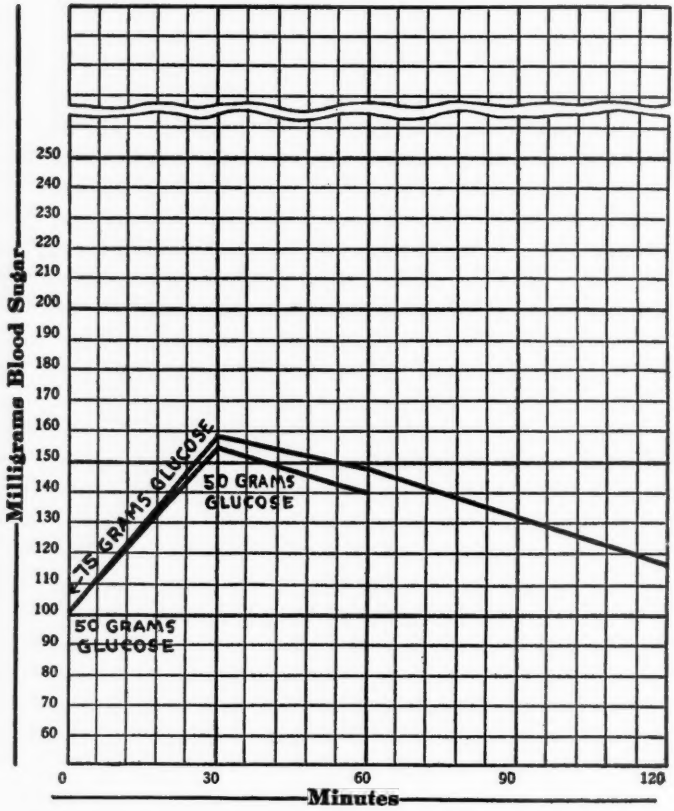
C

Thirty minutes after the ingestion of the second dose of glucose, collect (C) final blood and urine samples. The third urine container is given the subject for a sample of the urine next voided when a (post) sample is desired.

When the results of the foregoing procedure are plotted, the interpretation of the first part of the curve, i. e., the part which includes the original and the 30-minute samples, is exactly the same as the interpretation of the same part of the curve of the older procedure, and the same deductions are accordingly drawn from the blood and urine sugar values. The interpretation of the second part of the curve, i. e., the part which includes the 30 and 60- minute values, is not at all like that of the older test, and the following cases will serve to illustrate the criteria we

use for interpreting the results of one-hour two-dose tests. Chart 2 shows the average responses of ten normal males and ten normal females, ages 19 to 48, to one 75-gram and two 50-gram doses of glucose.

CHART 2



Responses of 10 normal males and 10 normal females (ages 19-48) to one 75 gram and two 50 gram doses of glucose. Note the decline in blood sugar after the second dose of glucose—no sugar in urine.

Starting at 100 milligrams it will be seen that the first parts of both curves are practically the same. The rise in blood sugar following 75 grams is usually a little higher than that following the 50-gram dose of glucose. This tendency is, however, by no means invariable because many cases show the same or a higher rise in the blood sugar after 50 grams than they do after 75 or even 100 grams of glucose. Such instances may be caused by more rapid and uniform absorption of the smaller amounts or concentrations of glucose, and illustrations of such reversals of the expected results are seen in several of the charts.

The second part of the one-hour two-dose curve, i. e., 30 and 60-minute values, indicates that normals respond to a second dose of glucose with a greater fall in blood sugar than that occurring during the same period after only a single dose of glucose. It is also characteristic of normals that there is no sugar in the urine. The typical criteria of normal responses to the one-hour two-dose test are, therefore:

1. *A fasting blood sugar within the normal limits of the particular blood sugar method employed.*
2. *Thirty-minute sample: A rise in blood sugar which does not exceed 75 milligrams.*
3. *Sixty-minute sample: The blood sugar is less, the same, or does not exceed the 30-minute sample by more than 5 milligrams.*
4. *All urine samples are negative to Benedict's qualitative copper test.*

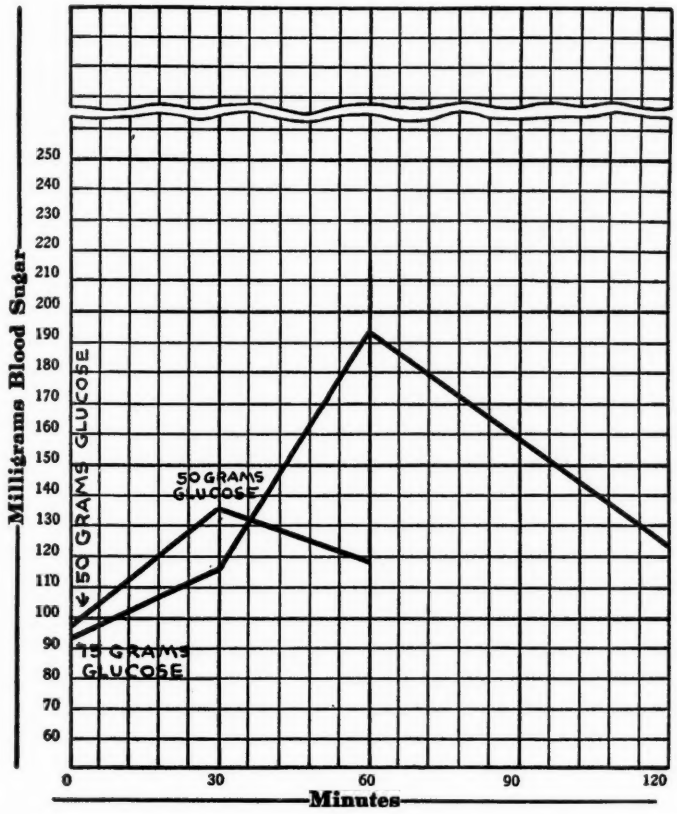
In the literature of alimentary sugar tolerance tests there is a good deal of discussion about the meaning of certain results which some consider significant of this or that condition, but which others consider atypical, doubtful or misleading. These questionable results typify some of the difficulties which detract from the diagnostic value of sugar tolerance tests. Thus, it is not at all unusual for results of sugar tolerance tests to take the form of curves having delayed peaks or curves with plateaux or curves which tend to flatness. We have had a number of these cases which gave doubtful or misleading responses to the older test

but perfectly typical and characteristic responses to the one-hour two-dose test. An obvious explanation of these discrepancies is offered by the differences observed after introducing glucose into the duodenum at various rates which Hale-White and Payne[17] have interpreted as meaning that the emptying rate of the stomach influences the glycemic period. Cases illustrating a delayed peak, a plateau and flatness in the atypical or doubtful curves given by the older test, but perfectly typical responses to the new test are seen in Charts 3, 4 and 5.

In this case it appears at 30 minutes that the increase in blood sugar is greater after 50 than after 75 grams of glucose. The same relationship is seen even more marked in some cases after taking 100 grams of glucose. The simplest explanation of this apparent paradox is the more rapid and uniform absorption of the smaller dose or concentration of glucose. In any event the older test in this case gives a doubtful response while the result of the new test is typically normal and does not raise any question of interpretation. As a matter of interest, this curve may be compared with the curve of similar contour by the older test seen in Chart 12 in which the action of epinephrin rather than irregular absorption is considered causative.

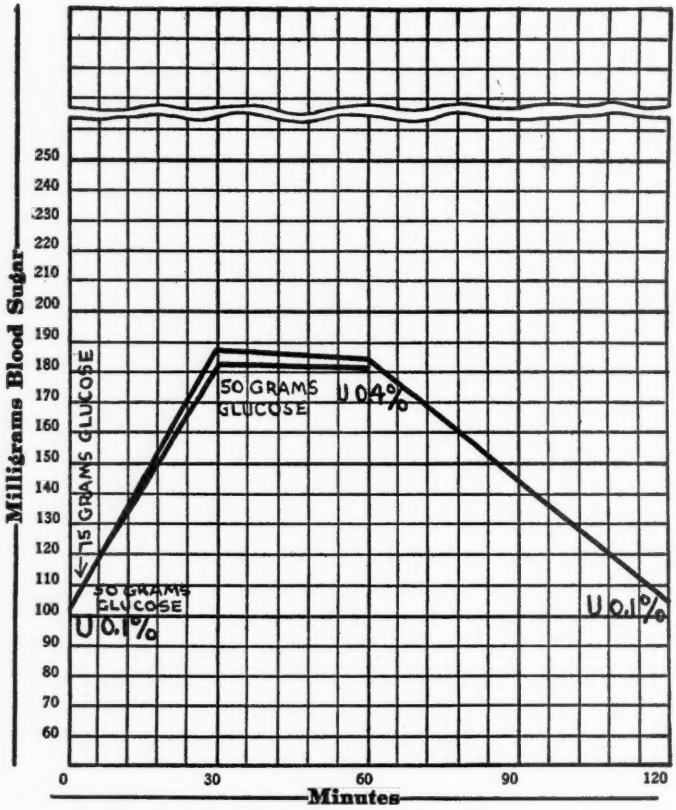
Of the cases giving plateaux curves by the older test some are normals and some alimentary glycosurias. The new test easily distinguishes between them by the sugar in the final urine and also by the height of the B or 30-minute blood sugar level which may run somewhat above normal in the alimentary glycosuria cases. The case in Chart 4 is as mild as any case of alimentary glycosuria we have seen, and as a matter of interest may be compared with the more outspoken case in Chart 10 which, it will be noted, does not have a plateau curve. Only relatively few normals and alimentary glycosuria cases give plateaux curves and therefore the plateau cannot be considered characteristic of any particular condition, but rather the evidence of some influence on the older test which does not affect the new one.

CHART 3



G. W. Case illustrates delayed peak curve of older test in a normal subject, probably due to delayed absorption of glucose. No sugar in any of the urine samples but blood sugar values of older test make the diagnosis doubtful. The result of the new test is typically normal.

CHART 4

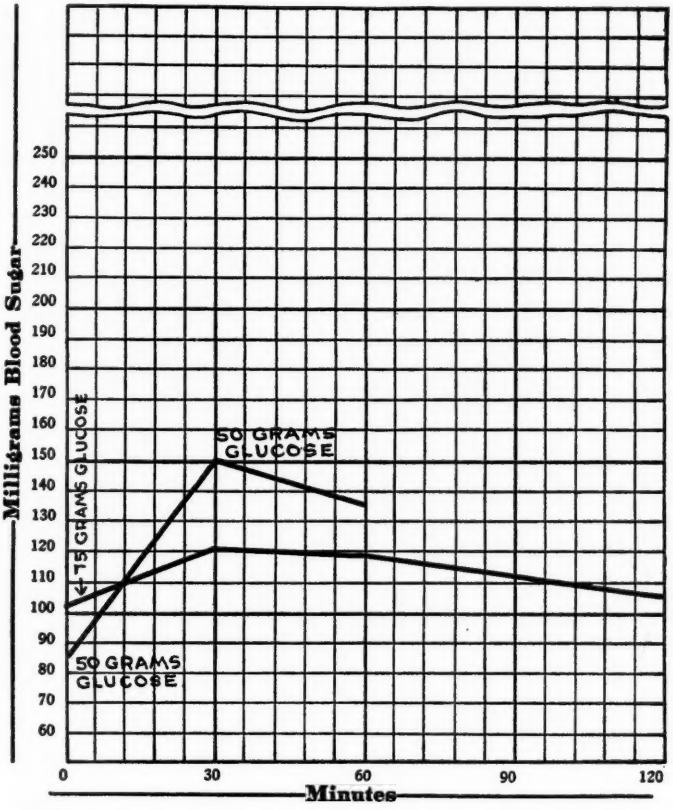


A. P. Case of Dr. Wm. Tilton has sugar in urine occasionally. Illustrates so-called plateau curve of old test. By the new test, the typical normal shape of the curve, the higher than normal level of the blood sugar, and the concentration of sugar in the final urine indicate a mild alimentary glycosuria.

Whatever significance some may attach to low flat curves, the carbohydrate tolerance of this case is perfectly normal. Many of the low flat curves we have seen were directly traceable to the

effects of glucolysis, but it is certain that glucolysis was not active in this case, not even in the slightest degree. It is also certain that there is no question of hyperinsulinism, a condition which some have offered as an explanation of certain low flat curves.

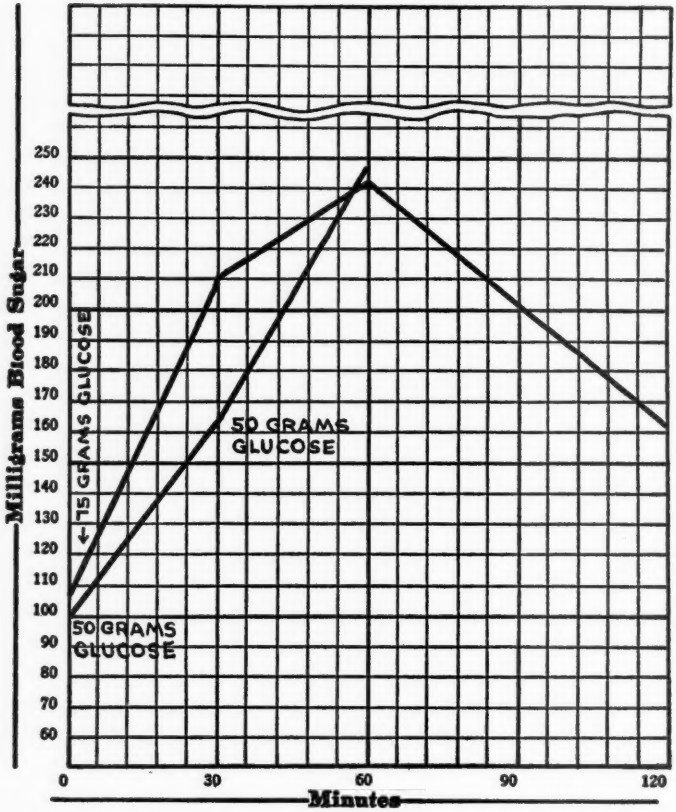
CHART 5



F. S. Case illustrates a typical flat curve of older test and typical normal result of new test.

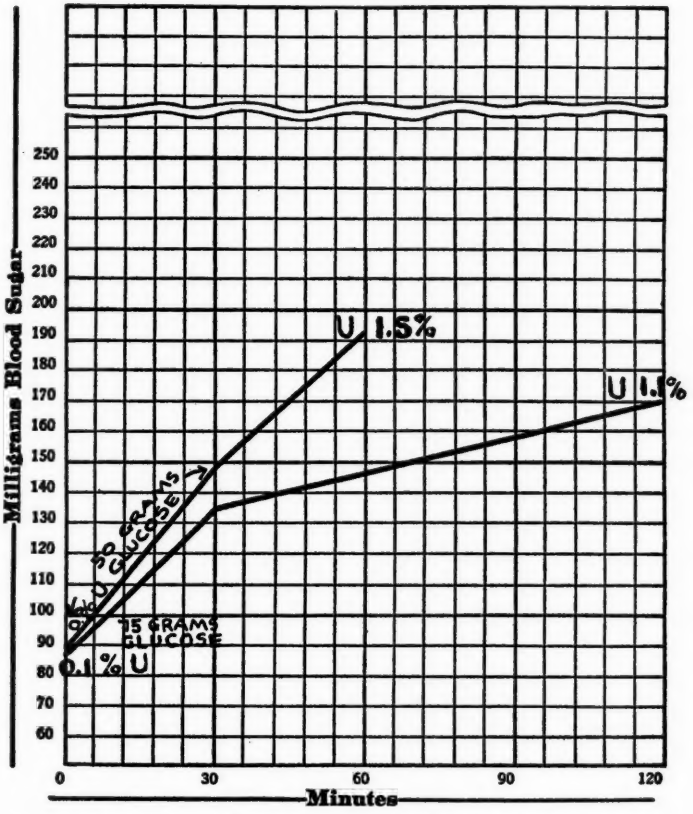
Unlike the preceding curves, those in Chart 6 represent the characteristic responses to both tests of a patient with mild diabetes. The responses of another mild diabetic in Chart 7 show a perfectly typical result of the new but an atypical result of the older test.

CHART 6



W. K. Case of Dr. W. R. Tilton. The steep climb of the blood sugar curve after the second dose of glucose is typical of diabetes.

CHART 7



Case of Dr. E. S. Dillon. Mild diabetes showing atypical result of older test due to irregular absorption and typical result of the new test.

A comparison of the older test curves at the end of the two-hour period suggests that the hyperglycemia of Dr. Dillon's patient is increasing when W. K.'s is decreasing. In W. K.'s case it is also evident that the rise in blood sugar after 75 grams of glucose is greater than that after 50 grams, as it logically should

be, while in Dr. Dillon's case the blood sugar rises higher after the lesser amount of glucose, contrary to theoretical expectations. This case therefore also illustrates how diabetics, like normals, give perfectly typical responses to the new test when responses to the older tests are atypical.

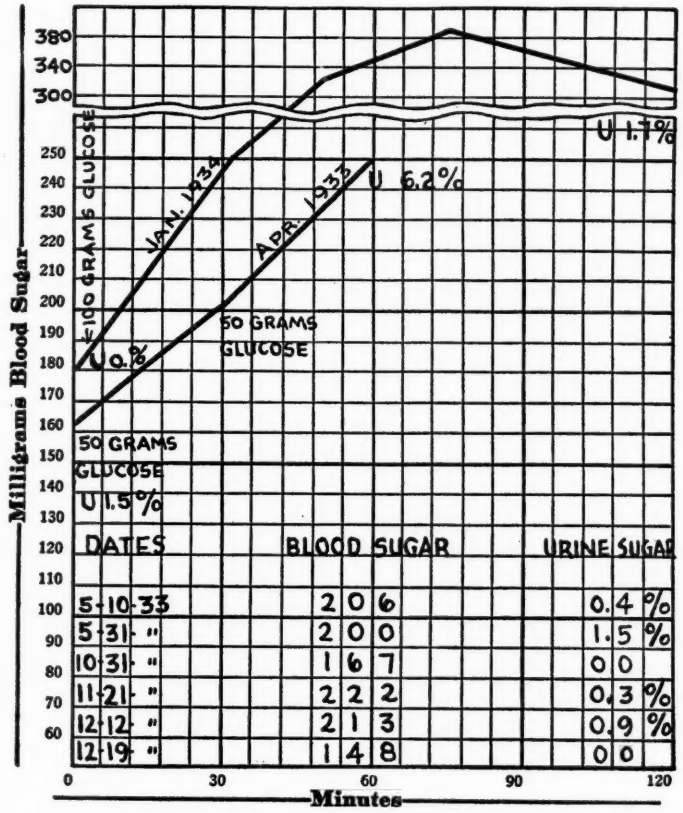
In the one-hour two-dose tests of both mild diabetics it is apparent that while the first parts of the curves are similar in trend to those of the older tests the latter parts which show the effects of the second doses of glucose are disinctly different. Here, the steep climb of the blood sugar after the second dose of glucose characterizes diabetes, and sharply distinguishes it from all non-diabetic conditions in accordance with the paradoxical law. In both cases it will also be noted that there is no sugar in the fasting urines, but sizeable concentrations in the samples voided after ingestion of glucose.

In more severe or advanced diabetes the responses to the one-hour two-dose test are in a general way the same as the responses of mild diabetes, only the sugar values of the blood and urine samples of the severer cases run higher. The tendency for the difference between milder and more severe diabetes to be quantitative is indicated in the curves of Chart 8 which show the results of old and new tests in a case of more severe diabetes.

From these illustrations it is evident that *the criteria for diabetes of the one-hour two-dose test are a more or less steep rise of not less than 10 milligrams of blood sugar in the 60-minute sample following the second dose of glucose and the relation of blood and urine sugar values to the severity of the disease.*

In some cases of diabetes the renal threshold for glucose changes from time to time as it does in non-diabetic derangements of carbohydrate metabolism. The case of F. S. shown in Chart 8 also illustrates such an instance. In some cases the urine shows little or no sugar even when the blood sugar values run very high. Such tendencies cannot be considered characteristic of any one particular condition but may be associated with complications or with derangements of carbohydrate metabolism secondary to clinical conditions other than diabetes.

CHART 8



F. S. Case of Dr. H. O. Mosenthal. Illustrates typical results of both tests in more severe diabetes, i.e., same criteria as mild diabetes but higher sugar values related to the severity of the disease. Case also illustrates tendency to high and changing renal threshold for glucose.

In our experience cases of renal glycosuria and pentosuria are wrongly diagnosed as diabetes oftener than other types of glysosuria, the pentosuria cases perhaps oftener than the renal

glycosuria cases. The mistake of diagnosing pentosuria as renal glycosuria is also not at all exceptional, and it should be noted that such mistakes seriously interfere with the attainment of an accurate statistical experience in these cases; something that is badly needed because our present experience is so extremely limited. It may also be pointed out that these mistakes can hardly be avoided unless the precaution is taken to identify the sugars found in urine because renal glycosuria and pentosuria give similar responses to tolerance tests.

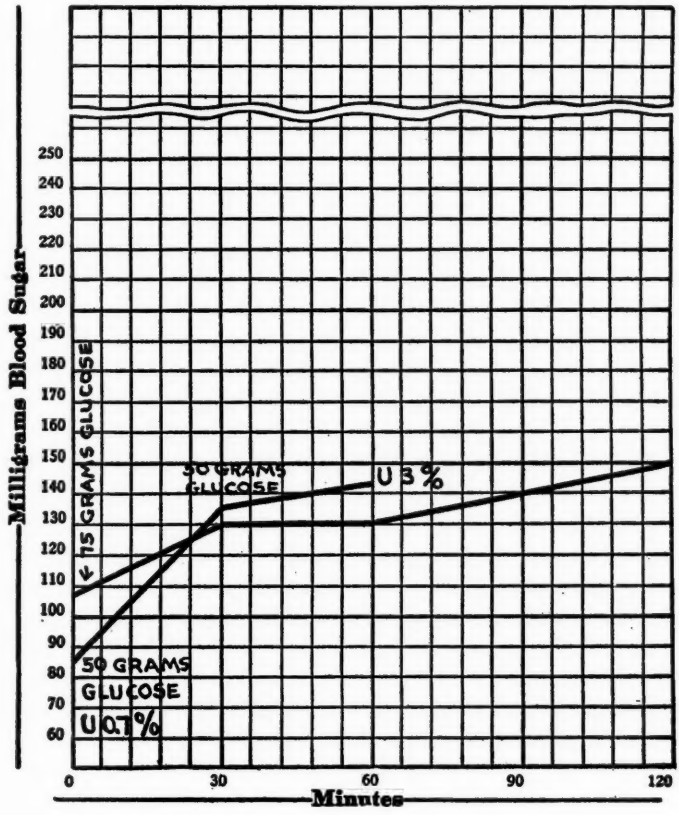
The diagnosis of renal glycosuria by the older methods rests upon criteria which are still subjects of considerable difference of opinion and practise. Perhaps the strictest criteria are those of Joslin and his associates, and Chart 9 shows the results of old and new tests on one of their cases of renal glycosuria (No. 2279 of Marble's report[18]). It should be noted that different blood sugar technics were employed in making these curves and that the new test was made several years later than the older.

In the one-hour curve it is of interest to find the blood sugar after the second dose of glucose in the borderland between normal and diabetic. While other cases of renal glycosuria have shown the same tendency, more cases show blood sugars like normals. The pentosuria cases behave in the same way, and it is, of course, characteristic for all urine samples to show sugar in both renal glycosuria and pentosuria.

The criteria of the one-hour two-dose test are the same in renal glycosuria and pentosuria, the only difference being the identification of the sugar found in the urine. These criteria are: Blood sugars which follow the normal course, or in any event never reach the diabetic level, and sugar in both urine specimens.

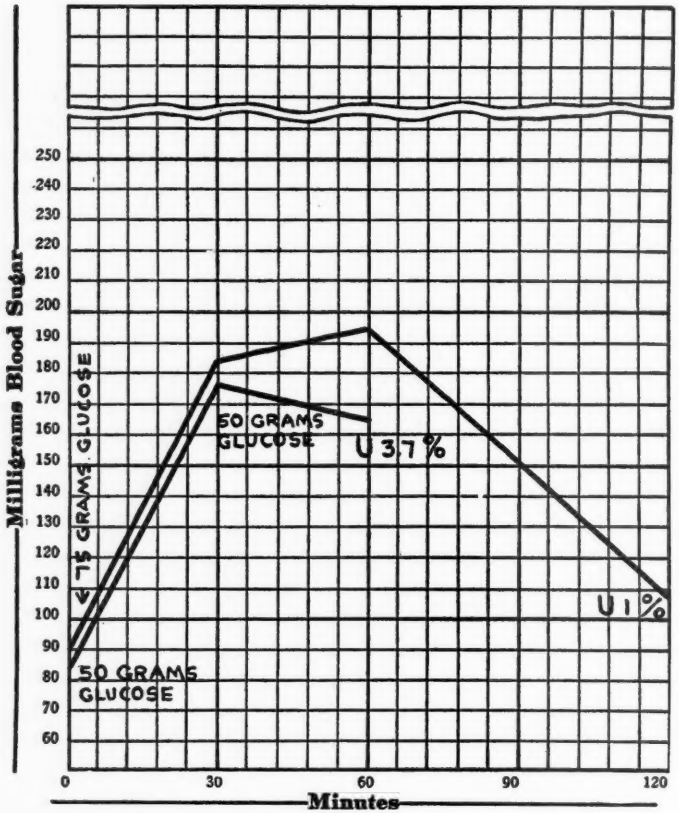
In addition to the persistent glycosurias, there are intermittent glycosurias which may or may not be diabetic. In these cases the sugar tolerance test is the only means at our disposal for distinguishing between diabetes and alimentary glycosuria.

CHART 9



In the alimentary glycosuria cases the fasting blood sugar is within normal limits like some of the diabetic cases. Following the first dose of glucose the 30-minute blood sugar in some of the alimentary glycosuria cases may remain within normal limits, but others may exceed them like the diabetics, sometimes very

CHART 10



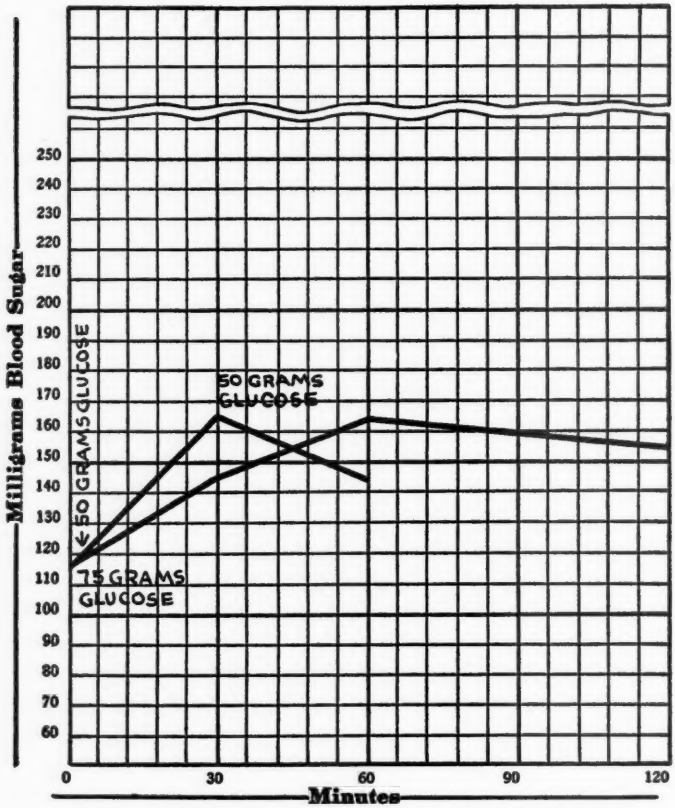
H. J. Case of Dr. P. V. Reinartz. In alimentary glycosuria there is no sugar in the fasting urine but definite amounts after ingestion of glucose. The new test sharply distinguishes between this condition and diabetes by the fall in blood sugar after the second dose of glucose instead of the rise characteristic of diabetes. Compare with Chart 4 showing a similar but milder case.

considerably. After the second dose of glucose, however, the difference between alimentary glycosuria and diabetes becomes very clear. The blood sugar distinguishes sharply between them because it rises characteristically in diabetes and falls in alimentary glycosuria like in normals. In both conditions the final urine samples show more or less sugar, depending upon the threshold of the individual. Chart 10, when compared with the diabetic illustrations in Charts 6, 7 and 8, shows the characteristic distinction between alimentary glycosuria and diabetes. It should also be compared with the milder case of alimentary glycosuria with plateau curve of the older test seen in Chart 4.

The criteria of the one-hour two-dose test for alimentary glycosuria are: A rise in the blood sugar, which is generally higher than the normal range in the 30-minute specimen after the first dose of glucose, and a stationary or declining blood sugar in the 60-minute sample after the second dose of glucose. The fasting urine does not show sugar but the final urine does, the amount depending upon the threshold of the individual.

While the foregoing conditions represent the usual clinical types of glycosuria in which the carbohydrate metabolism is of special interest, there are other conditions which secondarily or accidentally affect carbohydrate metabolism. Some of them are associated with glycosuria, generally intermittent in character; others are not associated with glycosuria. In some of the cases the disturbance of carbohydrate metabolism is temporary or trivial, as in colds and other infections, toxemias, menstruation, etc., and it is, of course, advisable not to make sugar tolerance tests at such times. In other cases the glycosuria depends upon more fundamental disturbances of carbohydrate metabolism which must be differentiated from diabetes, such as those associated with endocrine and psychic conditions. Chart 11 shows the effects of a thyroid disturbance on carbohydrate metabolism, and Chart 12 the effects on the curves of both tests of an emotional condition. In both cases the correct results of the new and the misleading results of the older test are evident.

CHART 11

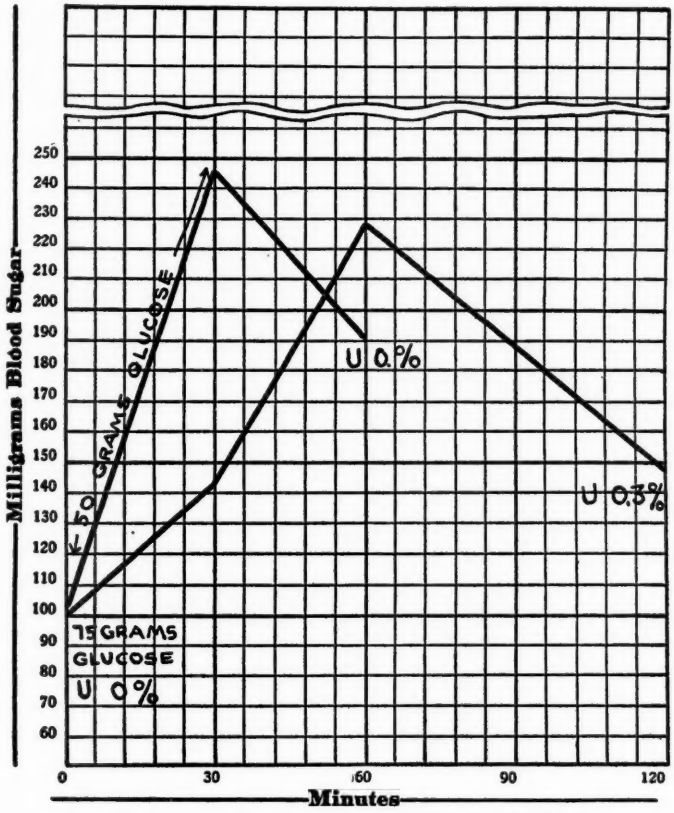


A. A. Case of Dr. M. K. Smith. Nervous and irregular menses. Under treatment for thyroid trouble sugar in urine occasionally. The new test definitely excludes a diabetic tendency but the older test does not.

In this case it will be observed that the curves made several days apart start from the same fasting blood sugar level and reach the same height suggesting that the carbohydrate tolerance of the subject was about the same on both occasions. It will also be

observed, however, that the curves give conflicting evidence because the curve of the older test points clearly to a diminished carbohydrate tolerance which the typical normal curve of the new test specifically denies. The subject is not diabetic.

CHART 12



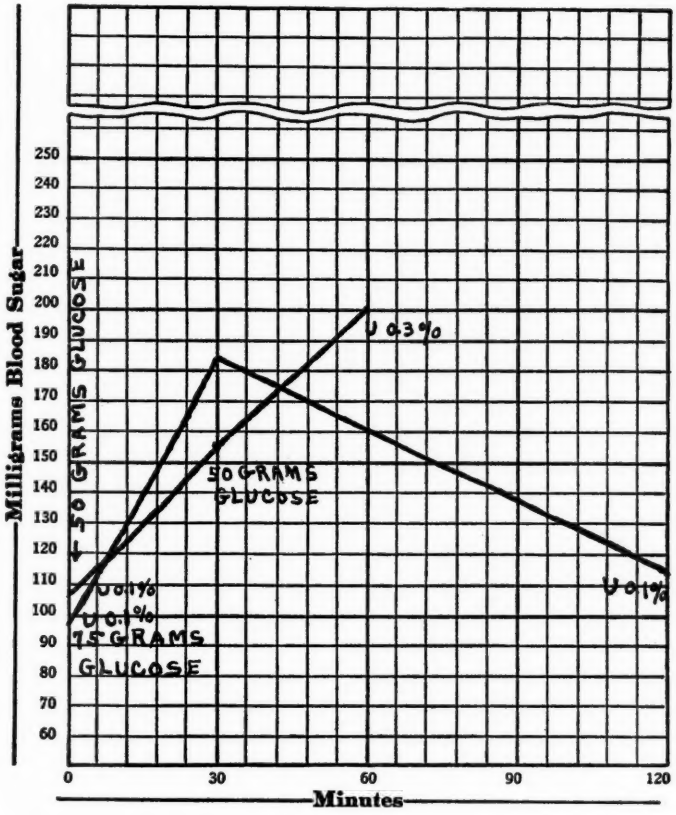
A. C. Case of Dr. W. R. Tilton. Illustrates psychic effects on sugar tolerance tests. In this case, emotional disturbance related to secret marriage and early pregnancy. The older test indicates a diabetic tendency but the new test does not.

Like the case shown in Chart 11, the curves made several days apart in this case also suggest that the carbohydrate tolerance of the subject was about the same on both occasions. Like in case 11, the curves in this case also give conflicting evidence. Thus, in this case too, none of the criteria for diagnosing diabetes by the older test are missing, while the new test emphatically denies diabetes or a diminished carbohydrate tolerance by the definite decline of the blood sugar after the second dose of glucose. In this case the only feature of the new test which marks a departure from normal is the abnormally high blood sugar in the 30-minute specimen after the first dose of glucose. This might be interpreted as a lag in insulin activation which sometimes makes itself evident in this way, but in this case it seemed more reasonable to attribute this effect to the action of epinephrin because this young subject was tested in a routine way along with other supposed normals who were doing their regular office work when we were collecting data on normals. Although her nervousness was noted, it was only after the surprising results were obtained that information concerning her true condition became available. The similarity of the responses to the older test of this and the normal shown in Chart 3 may also be observed.

An example of the use of the new test for life insurance selection is portrayed by the curves in Chart 13. On examination, about a year previously, the 40-year-old applicant had sugar in the urine, and a Home Office examination of a portion of the same sample was reported: Specific Gravity 1032, Sugar 5.5%. The older test was made ten days earlier than the new, and the curve negatives any diabetic tendency. The results of the new test, on the contrary, are typical of mild diabetes.

In conclusion, it remains to express our appreciation of the careful technical work of Miss Cecile Blacker, and to state that up to the present time not a single instance has been encountered in which the new test disagreed with the older when the results of the older test were satisfactory. On the other hand, the new test gave consistent and specific results in many cases in which the responses to the older test were doubtful or misleading.

CHART 13



SUMMARY

Some of the factors which influence the results of alimentary glucose tolerance tests have been discussed in connection with the development of a new procedure designed to avoid them.

A one-hour two-dose test based on Allen's paradoxical law of dextrose has been described and its diagnostic criteria illustrated

by applications of older and new tests to the more usual clinical types of glycosuria.

Besides its greater convenience, all of the available evidence indicates that results of the new test are more specific and reliable.

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DR. FRASER—We have now completed the program of the 45th annual meeting and I wish to take this opportunity to thank the Program Committee, the authors of the papers and the discussers of the papers personally as well as you gentlemen for your forebearance in listening to my croaking up here.

And now I have much pleasure in introducing the new President, Dr. Frederick Wells of the Equitable Life of Iowa. (Applause.)

DR. WELLS—Thank you, gentlemen. I have no speech, but I do want to thank you for the great honor of being elected your President, an honor both to me and to the company I represent. I am sure we are going to have a good meeting next year, and if I find it necessary to send out an S. O. S. call for assistance, I am sure that I will get a very gracious response, and if there are any of you who have any constructive criticisms as to our meetings, I should be very glad to have a letter from you.

DR. MUHLBERG—May I offer a resolution?

RESOLVED, That this Association express its thanks and appreciation for the very able manner in which Dr. Fraser has administered his office as well as the excellent quality of his program.

(The resolution was unanimously passed by a standing vote.)

DR. WELLS—Is there any further business?

DR. FRASER—Dr. Cragin has a motion.

DR. CRAGIN—I wish to call your attention to the fact that there is missing from this meeting a man who has been most active in the past and has presented many valuable studies, original and useful, for the Society. He has had a severe illness, but I am happy to learn that he is making a good recovery. I would

like to present a motion that it is the sense of this meeting that we send a communication, through the secretary, to Dr. Charles P. Clark, of the Mutual Benefit, expressing our regret at his absence, our joy in his recovery, our very best wishes for his future and our hope of his early return to our ranks.

(The motion was seconded and unanimously passed.)

DR. WELLS—Is there any further business, Mr. Secretary? If not, I will fulfill practically my first duty then of adjourning the meeting.

ADJOURNMENT

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS

Edwin H. Allen, M. D.	John Hancock, Boston, Mass.
George E. Allen, M. D.	National Life, Montpelier, Vt.
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Hiram H. Amiral, M. D.	State Mutual Life, Worcester, Mass.
Harry B. Anderson, M. D.	Imperial Assurance, Toronto, Ont.
Robert B. Andrews, M. D.	Massachusetts Mutual, Springfield, Mass.
Thomas D. Archibald, M. D.	North American, Toronto, Ont.
Edward McP. Armstrong, M.D.	Mutual Life, New York City.
Thomas M. Armstrong, M. D.	Philadelphia Life, Philadelphia, Pa.
William B. Aten, M. D.	Security Mutual, Binghamton, N. Y.
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Albert E. Awde, M. D.	Union Mutual Life, Portland, Maine.
Walter C. Bailey, M. D.	New England Mutual, Boston, Mass.
Henry A. Baker, M. D.	Kansas City Life, Kansas City, Mo.
Henry A. Bancel, M. D.	Mutual Life, New York City.
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Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.
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THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS OF AMERICA

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